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THE AMERICAN JOURNAL OF ROENTGENOLOGY AND RADIUM THERAPY

VOL. 51 JANUARY, 1944 No. 1

ROENTGENOLOGICAL TYPES OF PULMONARY LESIONS IN PRIMARY COCCIDIOIDOMYCOSIS

By JUSTIN R. COLBURN, *Major, M.C., A. U. S.*

DIAGNOSTIC roentgen criteria cannot alone be depended upon in differentiating primary coccidioidomycosis from other diseases it may simulate. However, scrutiny of roentgen evidence provided by studies of 75 cases seen during a recent "epidemic" indicates that fairly characteristic types of pulmonary lesions exist. When such lesions are observed by the roentgenologist, an opportunity for stimulating the search for corroborative laboratory and clinical data is presented.

HISTORICAL AND CLINICAL CONSIDERATIONS

For a review of the work leading up to and following the discovery of the primary stage of coccidioidal infection reference should be made to articles by Dickson and Gifford,[4] Smith[6,7] and Winn.[8] Pertinent facts are briefly summarized: The notable endemic areas for this disease are in California's central valleys and Arizona, and less prominently, New Mexico, Texas and Mexico. Caused by inhalation of dust-borne chlamydospores of the fungus in its vegetative stage, the disease characteristically produces acute or subacute symptomatology consisting of dry cough, chest pain, malaise, fever, weight loss and night sweats. Five to 20 per cent may develop erythema nodosum or multiforme. Gradual recovery is the rule, symptoms usually subsiding in two to six weeks, but resolution of the pulmonary lesions is often prolonged weeks after the patient is clinically well. In an extremely small percentage of cases the highly fatal chronic disseminated granuloma stage develops, apparently as endogenous "reinfection." Carter[1] has described these pulmonary and extrapulmonary lesions.

SOURCE OF CLINICAL MATERIAL

The cases reported consist of 75 soldiers admitted to the Station Hospital, Camp San Luis Obispo, California, between July 1 and August 15, 1942. This group is the subject of a paper by Goldstein and Louie,[5] in which the clinical and laboratory aspects of primary coccidioidomycosis are described. As therein indicated, the "epidemic" was associated with army maneuvers during the favorable dry, dusty season, affecting chiefly susceptible California nonresidents recently arrived in the endemic area. Symptoms, prominently respiratory, usually developed ten to fourteen days following exposure. Suspected cases were studied and grouped as "positive" when they presented all of the following criteria:

(1) Recent exposure to inhalation of spore-laden dust.

(2) No previous history of exposure in an endemic area.

(3) Incubation period within known upper and lower limits.
(4) Positive coccidioidin (intradermal) reaction.
(5) Positive complement fixation, or precipitin tests showing titers compatible with the stage and severity of disease present.

Additional diagnostic evidence presented by most cases included: pulmonary lesions demonstrated by serial roentgenograms, delayed resolution of such lesions, accelerated sedimentation rates, low grade fever, and moderately prolonged convalescence. Where sputum cultures were made, results were uniformly positive. Erythema nodosum or multiforme developed in 20 per cent of cases. Roentgenological follow-up studies were continued until pulmonary lesions were resolved or quiescent.

Serological tests and a number of sputum cultures were made available through the interest of Dr. Charles E. Smith, Department of Public Health and Preventive Medicine, Stanford University School of Medicine, San Francisco, California.

ROENTGENOLOGIC "TYPES"

Of the 75 proved cases of primary coccidioidal infection, 29, or 38.7 per cent, presented infiltrative fan-shaped densities extending from prominent hilar shadows. This "linear mottling," which may involve in varying degree any lobe or portion thereof, resembles bronchopneumonic inflammation with a minimum of consolidation (see Case I, Fig. 1). Resolution is always slow, ranging from fifteen to ninety days and averaging forty days. Thus, by serial roentgen examinations, the lesions are differentiated readily from those of ordinary bronchopneumonia.

Hilar densities without evidence of significant peripheral bronchial or parenchymal involvement constitute 18 cases, or 24 per cent of the group. In some instances circumscribed lymphadenopathic shadows are seen, particularly in the stage of early regression. Minimal lesions present only a loss of bronchovascular hilar detail and care must be exercised in order to avoid "over-interpretation" of technically deficient roentgenograms. The likelihood of —"missing"— parenchymal lesions not yet formed, or already resolved, should be borne in mind; in fact, considered seriously where unusual hilar shadows are encountered.

Peripheral lobular and sublobular exudates and infiltrates were found in 20 instances (26.6 per cent of cases). These fairly well circumscribed, rather homogeneous peripheral densities are practically always associated with abnormal hilar shadows but they lack the radiating peribronchial infiltrations seen in "bronchopneumonic" types. Involvement of lower lobes predominated slightly, and when upper lobes were affected the lesions of course resembled minimal "reinfection" tuberculosis. However, more rapid and complete resolution is a differentiating feature since this averaged thirty-seven days in our series, and since conspicuous fibrosis was rare.

Demonstrable cavitation occurred in 3 cases (4 per cent). One was no longer demonstrable in sixty days, and one healed in ninety-five days, coincident with clearing of associated minimal infiltration and hilar adenopathy. The other has persisted as a cyst-like excavation resembling those described by Winn,[8] and others.

Two cases (2.7 per cent) presented massive pleural effusion without evidence of parenchymal infiltration and these, having subsided slowly, showed considerable residual pleural thickening at ninety days. Significantly, both showed strongly positive complement fixation and precipitin tests, indicating possible dissemination of the infection, and in one case proof of dissemination was presented by the appearance of a granulomatous skin lesion from which the fungus was recovered. Of the first three preceding groups, 7 cases showed associated slight to moderate pleuritis, often regional and interlobar (see Case III,

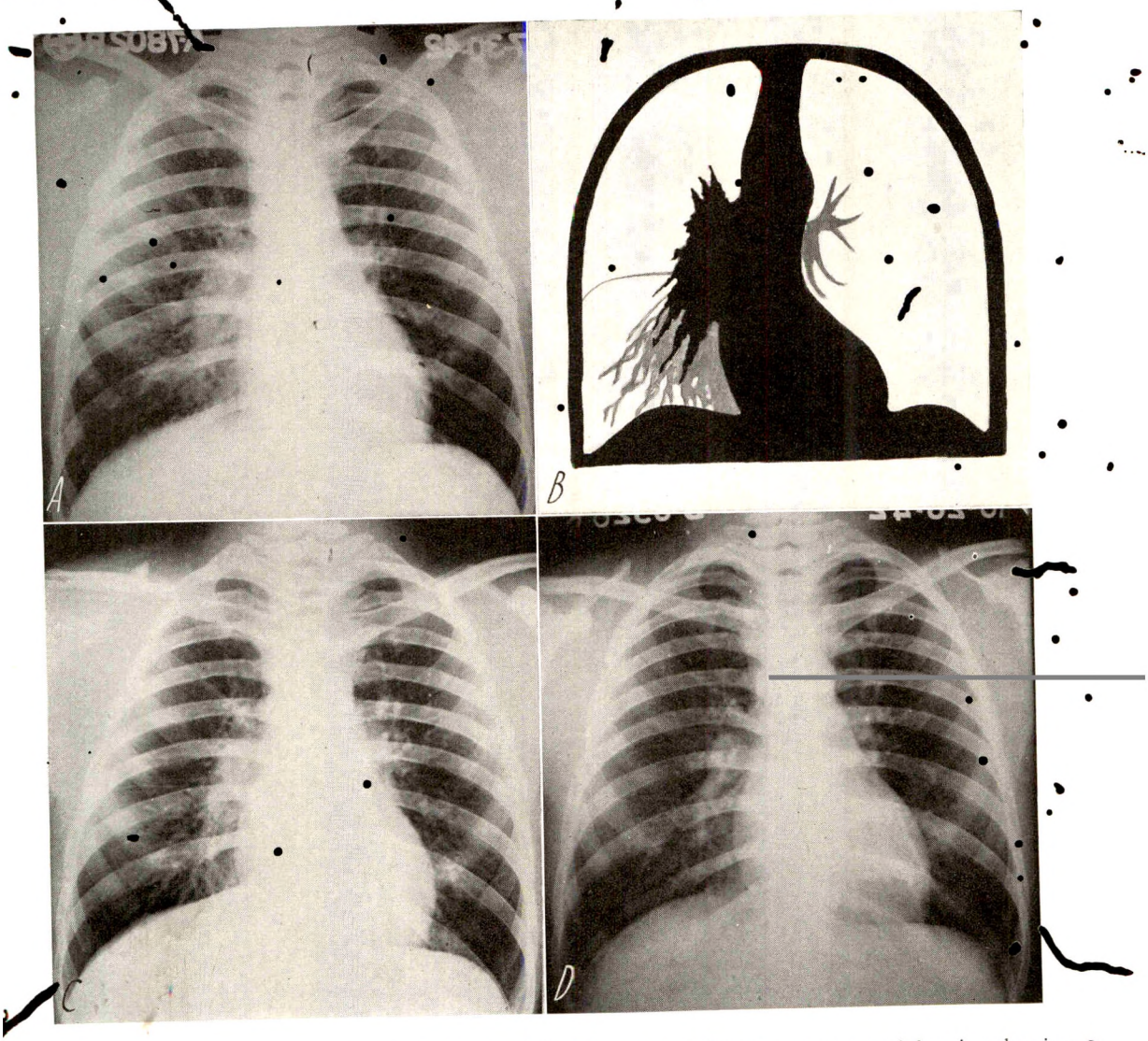


FIG. 1. Case 1. A. A. *Bronchopneumonic Type*. *A* and *B*, July 30, 1942. Roentgenogram and drawing showing enlarged, dense, confluent right hilar shadow, with linear and nodular mottling extending well into the right lower lung parenchyma. Minor fissure delineated. *C*, August 20, 1942. After three weeks considerable regression is evident, but infiltrates are still quite prominent and hilar shadow is accentuated. *D*, October 26, 1942. Accentuated bronchovascular markings remain, but shadows constituting the right hilum are more discrete.

Fig. 3). Clearing of such minimal pleural exudates was usually rapid and fairly complete.

Inconclusive roentgenographic evidence was considered to be present in only 3 cases (4 per cent) of this series. Obviously, only a portion of the primary infections resulting from the occasioned exposures are represented, since this group consists of soldiers whose respiratory and general symptoms were severe enough to interfere with performance of duty.

THEORETICAL CONCEPTS OF PATHOGENESIS

Production of a variety of pulmonary lesions by *Oidium coccidioides* is well explained by the possible state of balance of three important factors: individual resist-

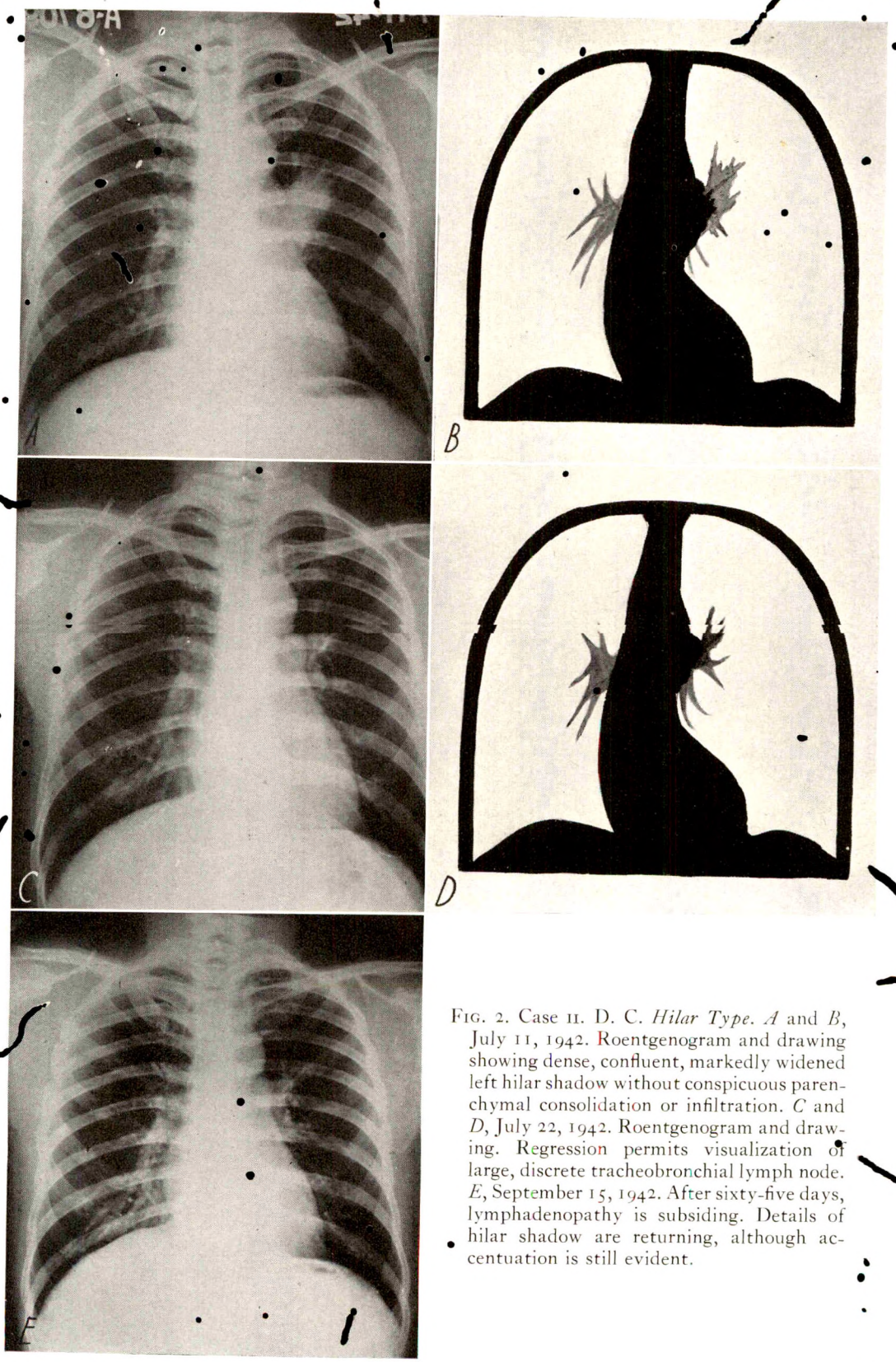


FIG. 2. Case 11. D. C. *Hilar Type*. *A* and *B*, July 11, 1942. Roentgenogram and drawing showing dense, confluent, markedly widened left hilar shadow without conspicuous parenchymal consolidation or infiltration. *C* and *D*, July 22, 1942. Roentgenogram and drawing. Regression permits visualization of large, discrete tracheobronchial lymph node. *E*, September 15, 1942. After sixty-five days, lymphadenopathy is subsiding. Details of hilar shadow are returning, although accentuation is still evident.

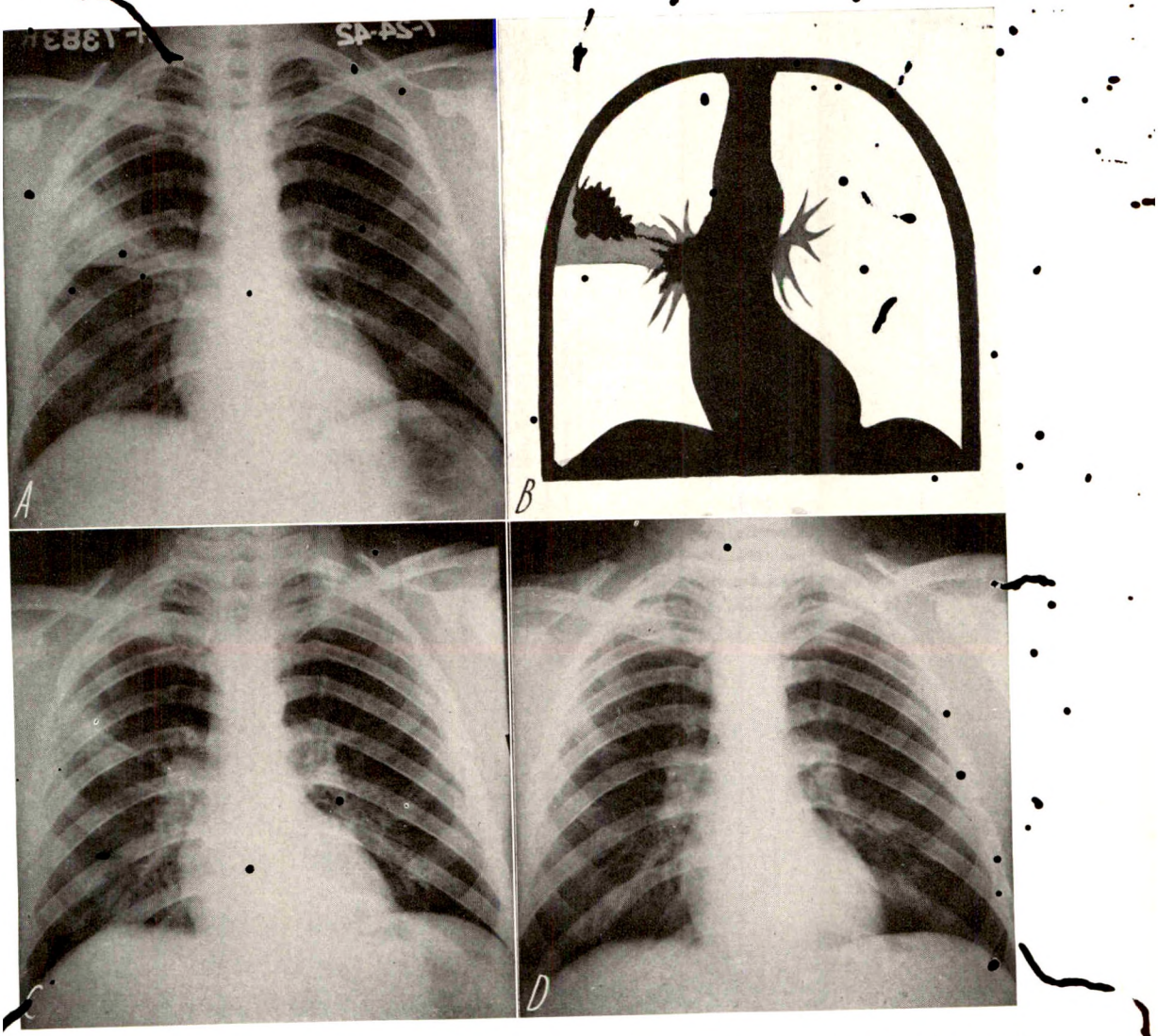


FIG. 3. Case III. O. R. *Peripheral Lobular Type*. *A* and *B*, July 24, 1942. Roentgenogram and drawing showing peripheral, circumscribed consolidation, right upper lobe, with associated regional localized effusion. Confluent hilar shadow. *C*, July 27, 1942. Rapid absorption of pleural exudate leaving lobular consolidation and moderately prominent right hilar shadow. *D*, August 19, 1942. Practically complete resolution of parenchymal consolidation. At twenty-six days only a few infiltrative strands and moderate hilar shadow prominence remain.

ınce, dosage of the infecting organism, and ıllergic status of the host. A fourth factor nay well be time, or the intervals of suc-:eeding repeated infecting doses in relation :o ascending allergy of pulmonary tissues. The interdependence of these factors would seem also to explain why manifestations of high allergy (coccidioidin positiveness and erythema nodosum) occur with variable time relations to the onset of symptoms (Smith[6]) and to the "onset" of exposure.

Application of these concepts to the le sions visualized is interesting, but not entirely satisfactory in the absence of further clinical and laboratory observations, such as bronchoscopy and serial coccidioidin, precipitin, complement fixation and sputum tests. However, such studies have not been available on a large case series. Pathological (autopsy) data are, for obvious reasons, lacking. The findings of Cronkite and Lack,[3] in experimental animals, can hardly be ap-

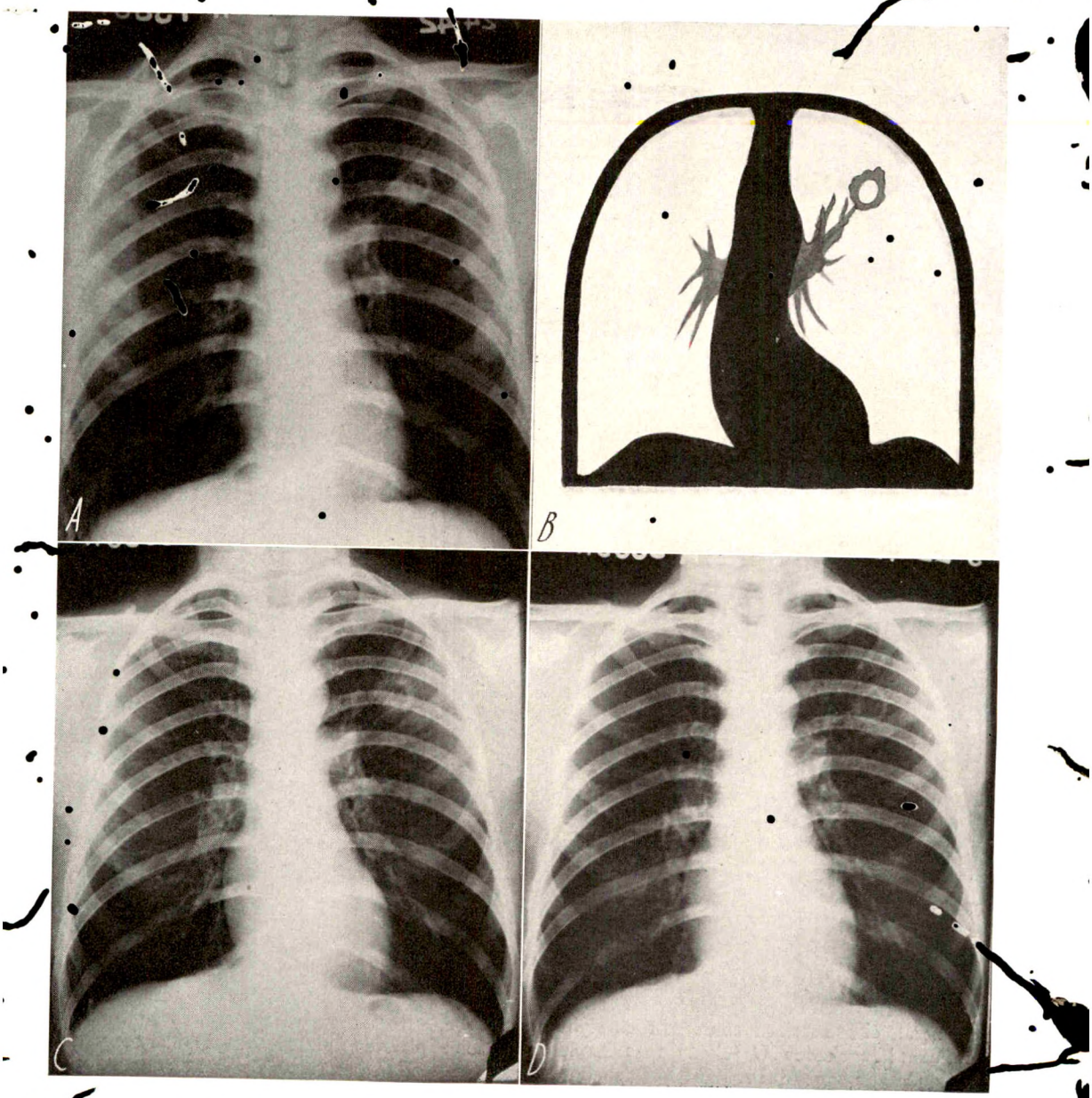


FIG. 4. Case IV. E. C. *A* and *B*, July 24, 1942. Roentgenogram and drawing showing cavitation in left upper lobe, with minimal surrounding inflammatory consolidation and infiltration. Hilar shadow only slightly prominent. *C*, September 7, 1942. Slight regression after thirty-five days. *D*, September 22, 1942. "Ballooning" of cyst-like cavity due to ball-valve bronchial obstruction. Cavity was only slightly smaller at ninety-five days.

plied to humans in view of evident differences in infecting doses and time intervals and in view of the relatively low sensitivity of guinea pigs to coccidioidin.

Despite the lack of such direct evidence, an attempt has been made to visualize the anatomical changes responsible for roentgen appearances. Well defined criteria are discussed separately:

1. *Loss of Hilar Shadow Detail; Slight General Increase in Hilar Density.* Probably due to initial invasion of mucosa and submucosa of primary and secondary bronchi with subacute inflammatory changes in

these tissues as well as in mild degree in the subjacent lymphatic structures.

2. *Confluence of Hilar Shadows with a Fringe of Accentuated, Poorly Delineated Bronchovascular Markings; Tracheobronchial Lymphadenopathy.* Continuation of the invasion of lymphatic structures provides obvious dense and more homogeneous hilar shadows, occasionally to the point where circumscribed enlarged lymph nodes are visible with accompanying evidence of often a lobule, respond to produce a fairly characteristic peripheral density. It is usually circumscribed, solitary (occasionally multiple and widespread), and associated with evidence of some degree of the above described hilar pathology.

5. *Pleural Exudates.* As in tuberculosis, the extent of pleural effusion has no regular relationship to the size and location of visible parenchymal and hilar lesions. Mechanical disturbance of lymphatic flow and

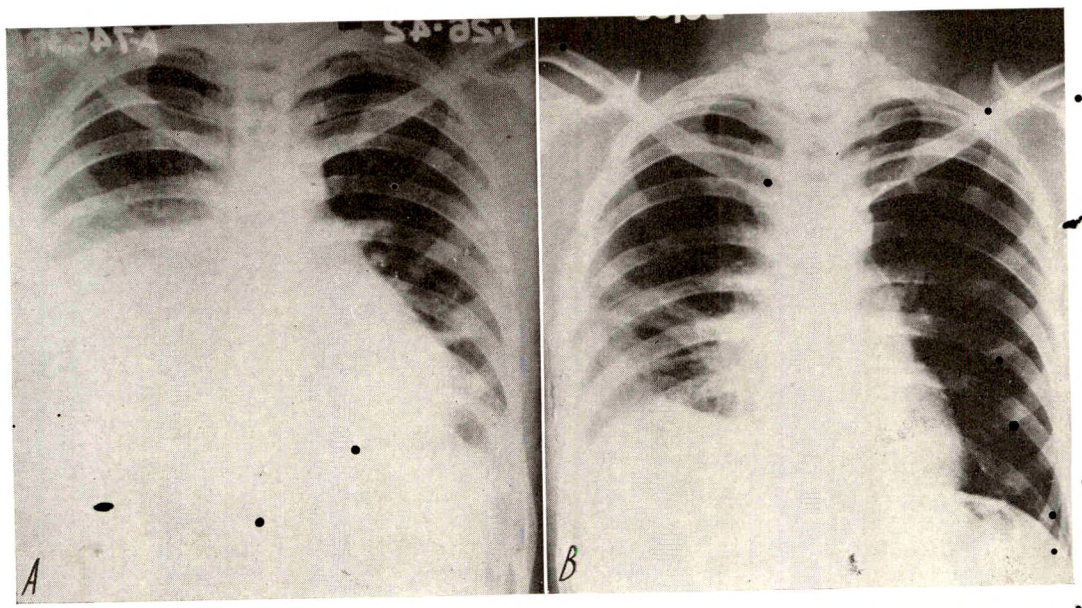


FIG. 5. Case v. S. C. *A*, July 26, 1942. Pleural effusion, right, with slight displacement of heart and mediastinum to the left. Parenchymal involvement, if any, obscured. *B*, October 12, 1942. Residual pleural thickening and right lower hilar prominence. Right diaphragm elevated and heart retracted to right. Patient developed metastatic granulomatous skin lesions.

peribronchial inflammatory exudate and cellular proliferation.

3. *Fan-shaped Extensions into Any Lobe of the Lungs; Linear and Nodular "Mottling."* Extension of the process into the tertiary bronchi and bronchioles produces the roentgen picture of bronchopneumonia. Alveolar involvement is limited and largely interstitial, and the peribronchial reaction is predominantly proliferative and cellular.

4. *Lobular and Sublobular Exudates and Infiltrates.* Depending upon the predominance of one or more factors, pulmonary parenchymal tissues in a localized area, tissue allergy, no doubt, are as important factors as the nearness of subjacent parenchymal lesions.

6. *Excavation of Pulmonary Tissues.* Small, cyst-like cavities with thin walls and little surrounding tissue reaction may persist long after infiltrates have cleared and after serological studies show that the infection is well localized. A bronchial factor was indicated in one of our cases by intermittent "ballooning" of a thin-walled cavity, and by its failure to fill when lipiodol was instilled into the area.

7. *Retarded Resolution of Pulmonary Le-*

sions. Indications of a predominantly proliferative reaction with a minimum of fibrosis are found in the characteristic course of the disease. Areas of exudation clear fairly rapidly (ten to twenty days) but infiltrates require thirty to ninety days and occasionally longer. Permanent fibrotic residues are not conspicuous.

8. *Calcification of Healed Lesions.* This has been observed by Cox and Smith[2] and is evidence of caseation of tracheobronchial lymph nodes during primary infection, with subsequent healing.

SUMMARY

The primary stage of coccidioidal infection is an acute or subacute respiratory infection varying greatly in severity of clinical manifestations, usually in accord with the extent of pulmonary lesions. However, because the initial lesion is evidently a low grade bronchitis, roentgenological findings are often inconclusive. In instances where sufficient numbers of coccidioides spherules attain the submucosa and lymphatics of the bronchi, their effects may be visualized roentgenologically. This was found to be true in 96 per cent of 75 cases hospitalized because of primary infections. Progressive involvement of distal bronchial and peribronchial tissues may produce roentgen shadows identical with those found in bronchopneumonia, or in tuberculosis when upper lobes are involved. Nearly as often, circumscribed lobular and sublobular exudates and infiltrates with associated dense hilar shadows appear. Regional and massive pleural exudates are fairly common, and cavitation may occur and persist despite apparent clinical recovery. The most constant finding is hilar shadow confluency, widening and increased radiopacity. The most valuable diagnostic roentgen criterion is delayed but fairly complete resolution, placing the disease midway between "acute" and "chronic" in the scale of pulmonary inflammations.

CONCLUSION

Roentgenological evidence in 75 cases of primary coccidioidomycosis is classified with the idea of establishing diagnostic criteria. Common types of pulmonary lesions are described and possible pathogenesis suggested. The importance of hilar shadow changes and of observations during resolution are emphasized.

Photographic reproductions by T/3 Louis C. Roberts, Assistant Chief Technician, X-Ray Service, Station Hospital, Camp San Luis Obispo, California.

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ACUTE PHOSGENE POISONING

ROENTGEN FINDINGS IN THE LUNGS: CASE REPORT

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ALTHOUGH lung changes in acute phosgene poisoning are undoubtedly quite common, published roentgenograms of this condition are very few.[1] The following case report demonstrates these changes and is, as well, of general interest at this time.

CASE REPORT

M. B., aged twenty-eight, a member of the Chemical Warfare Service, was, on the afternoon of May 20, 1943, decompressing a "sniff-set" containing phosgene. (A "sniff-set" is a small bottle of gas used for training purposes by the Chemical Warfare Service.) He was exposed to the gas for several seconds. The only immediate effect was momentary coughing. This was followed by headache and slight nausea, progressive in nature, until midnight, when coughing became more severe. Weakness, nausea and vomiting and tickling sensation in the chest became more marked at this time. He was admitted to the Station Hospital, New Orleans Port of Embarkation, New Orleans, Louisiana, at noon, May 21, 1943, twenty-two hours after exposure to the gas.

Physical examination on admission showed an acutely ill and apprehensive white male with distended neck veins and considerable cyanosis of the nail-beds and lips. Pulse was 132, respirations 42 per minute, temperature 100.5°F., blood pressure 120/82. The white blood count was 12,650, 73 per cent segmental forms, 24 per cent lymphocytes. Red blood cell count was 4.5 million. Examination of the chest showed numerous large moist râles on both sides, indicative of a "wet" lung.

The patient was placed in an oxygen tent; sedation was given and 50 per cent glucose intravenously. Improvement was very slow for the first few days. Sulfadiazine, grams 1 q 6 h, to a total of 15 grams, was administered in order to prevent secondary bronchopneumonia. The generalized râles disappeared gradually. By the fourth day they were localized to the left base. They were gone by the tenth day. The general condition of the patient ran parallel to the lung findings. Oxygen was discontinued by stages, beginning on the fourth day.

Roentgen examination immediately on admission (Fig. 1) showed a diffuse, soft, bilateral mottled infiltration, interpreted as edema of the lungs, due to irritant gas. (Figures 1, 2 and 3 were taken with portable, supine technique.)

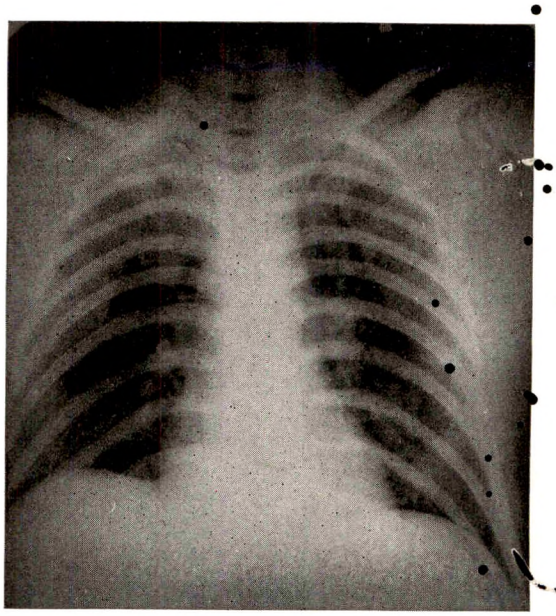


FIG. 1. Roentgenogram taken twenty-two hours after exposure to gas shows a diffuse, soft, mottled infiltration and thickening of the lung markings bilaterally, interpreted as pulmonary edema due to irritant gas.

Forty-eight hours later (Fig. 2) this had decreased slightly in extent except at the left base. Seventy-two hours after admission (Fig. 3) the process was shown to be localized mainly at the left base. There was still a slight generalized infiltration. Six days after admission (Fig. 4) there was still a slight residual infiltration at the left base and thickening of the root markings in this area.

Lipiodol study (Fig. 5) done on July 1, 1943, showed no change in the visualized bronchial tree.

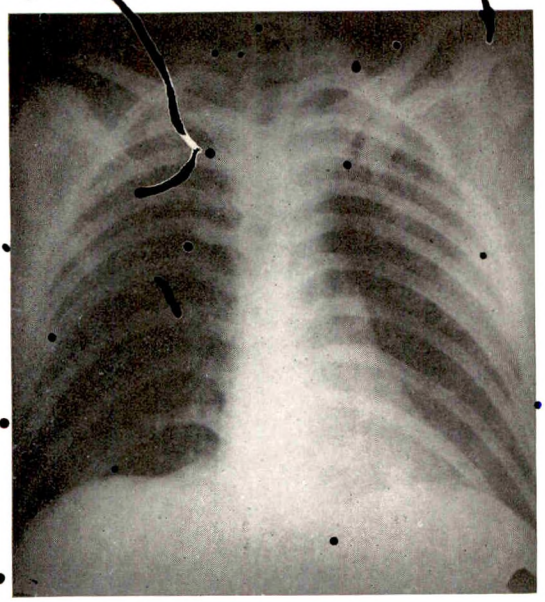


FIG. 2. Roentgenogram taken seventy hours after exposure to gas shows slight decrease in the extent and severity of the process.

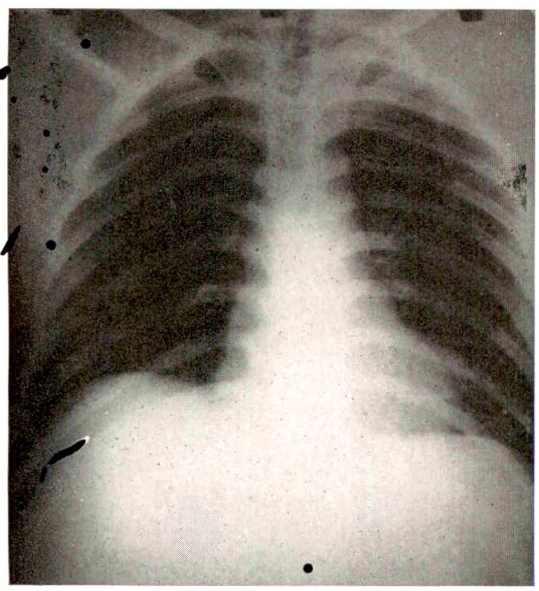


FIG. 3. Roentgenogram taken ninety-four hours after exposure to gas shows the localization of the process at the left base.

COMMENT

The outstanding pathological feature in the lungs in acute phosgene poisoning is pulmonary edema.[2] This is often the most serious clinical aspect of the condition. Serial roentgenograms, as demonstrated in

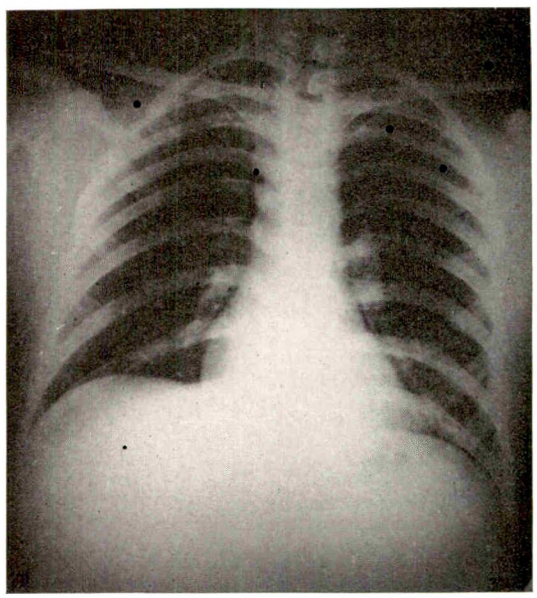


FIG. 4. Roentgenogram taken seven days after exposure to gas shows residual infiltration at the left base and thickening of the lung markings in this area.

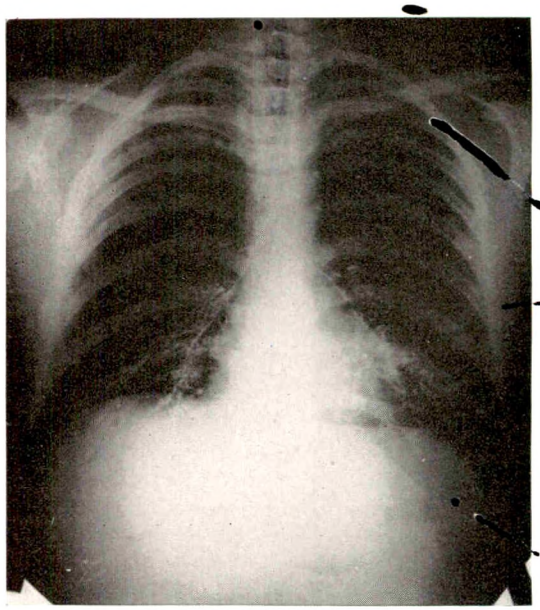


FIG. 5. Roentgenogram taken six weeks after exposure to gas, with lipiodol instillation, shows no demonstrable abnormality of the visualized bronchial tree.

this case, show the extent and severity of the process and the results of treatment.

Other gases, such as chlorine, diphosgene, nitric oxide and chloropicrin are lung irritants and may produce similar changes.[3,4] Different physical properties of some gases, however, result in differences in the nature and distribution of the pathology in the respiratory tract.[3,4]

Residual damage to the lungs is said to occur in some cases of phosgene poisoning.[2] Roentgenologically, this case showed complete clearing of the acute process. The patient's only complaint at last examination was an afternoon cough. However, secondary changes may be demonstrable at a later date.

Lipiodol study showed no change in the visualized bronchial tree at six weeks. This study was not done sooner because the added insult to the lungs at such a time might have done considerable damage. Later study with lipiodol is indicated.

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CHOLECYSTOGRAPHY AND JAUNDICE

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REVIEW of the literature reveals a surprising divergence of opinion as to the safety and value of cholecystography in patients with jaundice.

Ottenberg and Abramson[15] in 1925 warned that the administration of gallbladder dye might be dangerous in the presence of liver disease.

In the same year Lange[10] reported that uniformly a gallbladder will fail to visualize in the presence of jaundice irrespective of whether the jaundice is obstructive or infectious. No untoward effect due to the administration of dye in jaundice was noted.

In 1926 Fried and Whitaker[7] showed that even after damage of dogs' liver by chloroform anesthesia, the drug could be used in amounts equivalent to four times the human dose. With extreme liver damage no shadow was obtained.

Graham, Cole, Copher and Moore[8] in 1928 believed that patients having acute liver infection or common duct obstruction should not be submitted promiscuously to cholecystography, although actually injected cases showed no greater percentage of reaction. They also stated that they had found that the gallbladder can usually be visualized in cases of jaundice, irrespective of its cause. Their practice was to omit cholecystography as it may be wholly deceptive.

Rudisill,[16] in 1930, reported the results of examination of 10 patients with jaundice by the intravenous injection of 3.5 grams of sodium tetraiodophenolphthalein. He noted no unusual reaction and advised intravenous cholecystography as a possible means of helping to differentiate causes of jaundice. In 7 of 8 cases of infectious jaundice he obtained successful visualization. One of the other 2 cases was deeply jaundiced due to a common duct stone; nevertheless, a gallbladder full of stones was visualized. In the last case no shadow was seen. Autopsy showed cancer of the common duct.

Walsh and Ivy,[18] in 1931, reported the results of two experiments: (1) Common duct obstruction was produced in dogs after intravenous visualization of the gallbladder. In spite of daily fatty meals the shadow persisted for at least two weeks. (2) After production of common duct obstruction in dogs, retarded and faint gallbladder visualization was obtained after intravenous injection of tetraiodophenolphthalein. The toxicity of the drug was apparently not increased by the jaundice.

Also in this year Nord[13] demonstrated that it was often impossible to obtain a normal cholecystogram in the presence of acute hepatitis, particularly when the degree of icterus was increasing. There appeared to be a parallelism between the ability of the liver to excrete bile and dye.

Blomström and Sandström,[2] in 1932, showed that bromsulfalein retention of over 40 per cent indicated sufficient liver damage to prevent gallbladder visualization.

Foley,[5] in 1933, reported 29 cases, all but one by the oral method. No untoward reactions were noted. He concluded that visualization of the gallbladder in the presence of jaundice means an intrahepatic cause for jaundice, while no shadow is of no diagnostic significance. Of this series, 16 cases were medical and in 11 of these the gallbladder was visualized. The other 13 cases were surgical (i.e., common duct obstruction due either to stone or cancer) and none of these was visualized.

Also in 1933 Ferguson and Palmer[4] in a series of 1,398 cases of good visualization found among them: 1 case of atrophic cirrhosis; 8 cases of catarrhal jaundice; and 3 cases of neoplasm of the liver.

McWhirter,[11] in 1935, stated that chole-

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TABLE I

SUMMARY OF 50 UNSELECTED CASES OF JAUNDICE EXAMINED BY CHOLECYSTOGRAPHY

Patient	Age	Sex	Diagnosis	Degree of Jaundice	Cholecystographic Findings
			INCREASING JAUNDICE		
L. W.	48	F	Probable cholelithiasis with common duct obstruction	3/15/35— 10; subsequent clinical increase	No shadow (3/21/35)
E. C.	55	F	Common duct stone; operated 8/19/38	8/15/38— 13.2; subsequent clinical increase	No shadow (8/13/38)
W. S.	46	M	Probable common duct stone	1/ 3/37— 10.5 1/12/37— 40 1/19/37— 30.5	No shadow; 4 day examination (1/17/37)
H. F.	58	M	Diagnosis cancer of pancreas; operated 5/2/35	4/25/35— 32 5/ 1/35— 50	Faint shadow; some emptying (4/22/35)
H. K.	59	F	Common duct stone; operated 11/9/38	10/27/38— 51 11/ 1/38— 72 11/ 7/38— 50	No shadow (10/29/38)
S. W.	62	M	Primary cancer of liver; no common duct obstruction; autopsy 7/27/38	7/12/38— 13 7/19/38— 36 7/26/38— 50	Fairly dense shadow with emptying (7/14/38)
K. K.	38	F	Chronic cholecystitis and hepatitis; no stones; operated 3/5/36	12/30/35—250 1/ 4/36—125 1/18/36— 80 2/15/36—115	Faint shadow; some emptying (2/1/36)
M. F.	61	F	Atrophic cirrhosis of liver at autopsy 7/12/35	7/ 2/35—142; subsequent clinical increase	No shadow (7/7/35)
F. K.	58	M	Cancer of common duct; operation 3/16/39 (cholecystitis and cholelithiasis)	3/ 7/39—115 3/17/39—158	No shadow (3/16/39)
H. G.	75	M	Cancer of liver; operation 2/11/39	1/31/39— 55 2/ 5/39—101	No shadow (2/3/39)
W. [illegible]	52	M	Duodenal ulcer at roentgen examination	10/20/39— 20 10/23/39— 10 10/30/39— 20	No shadow (10/26/39)
J. N.	71	M	? Gained 15 lb. after discharge from hospital; still alive 4/1/39	11/11/38— 59 11/18/38— 56 11/26/38— 78	No shadow (11/21/38)
M. K.	67	F	Cancer of common bile duct; operation 7/19/39	6/30/39— 66 7/13/39— 82 7/29/39—120	No shadow (7/3/39)
B. B.	47	F	Cancer of pancreas; operated ated 2/18/35	Deeply jaundiced; increasing clinically	No shadow (3/15/35)
			DECREASING JAUNDICE		
G. M.	40	F	Clinically spontaneous passage of common duct stone	8/ 1/36—100 10/ 2/36— 41 10/23/36— 26	No shadow (10/1/36), (10/24/36)
P. L.	68	M	Catarrhal or toxic jaundice; alive and well 5/19/40	10/10/38—200 10/20/38—166	Very small faint shadow with emptying (10/13/38)

TABLE I—*Continued*

Patient	Age	Sex	Diagnosis	Degree of Jaundice	Cholecystographic Findings
			DECREASING JAUNDICE		
W. B.	[illegible]	M	Clinically acute toxic hepatitis; alive and well 1/30/40	9/21/38—250 10/24/38— 52	Small faint shadow (9/27/38)
S. C.	7	M	Diagnosis clinically catarrhal jaundice; later generalized lymphadenopathy without jaundice	4/27/38— 80 5/10/38— 20	Good shadow with emptying (5/ 4/38)
G. F.	11	M	Clinically catarrhal jaundice	5/17/38— 40 5/28/38— 8	Fairly dense shadow (5/26/38)
H. S.	11	F	Clinically catarrhal jaundice	4/ 2/38— 45.5 5/11/38— 23	Good shadow (5/14/38)
K. C.	34	M	Clinically catarrhal jaundice; alive and well 4/25/37	5/ 7/36—227 5/27/36— 29 6/ 2/36— 22	Good shadow (6/1/36)
A. D.	42	F	Clinically catarrhal jaundice; alive and well 11/10/35	2/15/35—250 3/ 2/35— 58	Good shadow (3/8/35)
M. G.	42	F	Clinically infectious jaundice; died; no autopsy	9/28/36— 80 10/16/36— 50	No shadow (10/10/36)
H. H.	47	M	Clinically catarrhal jaundice; alive and well 11/21/37	10/10/36—172 10/17/36—153 10/29/36— 77 11/23/36— 20	Faint shadow; some emptying (10/22/36)
P. G.	65	M	Clinically spontaneous passage of common duct stone	10/ 6/38— 27 10/14/38— 11	No shadow (10/10/38)
B. A.	52	F	Cholelithiasis and cholecystitis; operated 7/12/37	6/16/37— 57 6/21/37— 24 7/ 1/37— 17	No shadow (6/25/37)
A. B.	73	F	Probable inflammatory lesion of head of pancreas and liver; gallbladder negative; peritoneoscopy 11/1/38; died 2/2/39; no autopsy	10/10/38— 50 10/21/38— 33	No shadow (10/17/38)
A. K.	65	F	Common duct stone; operated 12/16/37	12/ 6/37—100 12/14/37— 66.5	No shadow (12/8/37)
J. B.	67	M	Cancer common duct; operated 10/28/39; (cholecystogastrostomy) (cholelithiasis); autopsy 11/2/39	10/19/39—165 10/26/39—156	Faint shadow with opaque stones (10/26/39)
E. G.	47	F	Clinically spontaneous passage of common duct stone	2/15/38— 10; subsequent decrease	Faint large shadow; several small stones (2/17/38)
J. T.	59	F	Clinically chronic cholecystitis and hepatitis; acute cholangitis.	6/ 1/35— 31 6/ 5/35— 16 6/15/35— 12	Very faint shadow (6/15/35)
R. W.	64	F	Cholelithiasis; operated 7/28/38	7/13/38— 42 7/18/38— 14.5 7/23/38— 14	Faint shadow with stones; no emptying (7/26/38)
A. W.	23	F	Cholelithiasis, cholecystitis	11/12/34— 20 11/23/34— 7	Very faint shadow with probable stones (11/19/34)
M. McC.	42	F	Cholecystitis, cholelithiasis; operated 5/3/34	4/24/34— 15; subsequent decrease	No shadow (4/26/34)

TABLE I—*Continued*

Patient	Age	Sex	Diagnosis	Degree of Jaundice	Cholecystographic Findings
			DECREASING JAUNDICE		
J. H.	71	M	Cholelithiasis, common duct stone	7/31/39— 20 8/ 4/39— 18 8/10/39— 11	No shadow (8/4/39) (gallstones in gallbladder and duct system)
O. Z.	46	M	Probable cirrhosis of liver; died 8/1/39; no autopsy	4/ 8/38—200 4/14/39—176 5/ 3/39—165	No shadow (4/13/39)
G. F.	45	M	Common duct stone; removed at operation 6/28/39	5/17/39— 50 5/25/39— 37.5 6/10/39— 20	No shadow (5/22/39); (choledochogram showed common duct stone) (6/27/39)
H. F.	62	M	Acute and chronic cholecystitis; no stones at operation 6/7/39	5/20/39— 44 6/ 1/39— 11	Very faint shadow (6/3/39)
F. E.	30	M	Clinically catarrhal jaundice	9/ 1/34— 75 9/12/34— 24	Good shadow (9/19/34)
R. S.	59	M	Autopsy 6/20/39; cancer head of pancreas with generalized carcinomatosis	8/13/37—143 8/17/37—154 9/21/37— 22 1/15/38—100 6/ 3/38— 80	No shadow (8/5/37)
E. J.	37	F	Probable spontaneous passage of common duct stone	4/12/40— 80 4/22/40— 26.6 4/29/40— 20	Faint shadow with non-opaque stones (4/25/40)
J. P.	31	M	Common duct stone; operation 12/15/38	12/ 3/38— 20 12/17/38— 6.6	No shadow (12/1/38 and 12/13/38)
V. D.	29	F	Cholelithiasis; operated 4/22/39	3/17/39— 22 3/27/39— 8	No shadow (3/23/39); calcified stones
E. D.	45	F	Cholecystitis; no stones; operated 2/11/39	2/ 7/39— 25 2/ 9/39— 18	No shadow (2/8/39)
M. S.	49	F	Probable common duct stone; refused operation	2/14/39—100 2/24/39— 96 3/20/39— 55 3/27/39— 33	No shadow (2/20/39)
M. H.	66	F	Cholecystostomy in 1933 for cholelithiasis; common duct stone; removed at operation 3/2/40; cholecystectomy 6/27/40; no complaints	2/ 3/40— 22.5 2/23/40— 10 4/23/40— 12 5/ 2/40— 9	No shadow (2/5/40) (cholangiogram 3/26/40 showed common duct stone)
J. C.	6	M	Hepatitis due to sulfapyridine; alive and well 6/4/40	3/22/39— 66 4/ 1/39— 40 4/20/39— 20	Good shadow (4/13/39)
J. C.	54	F	Common duct stone; cholecystectomy and removal of common duct stone 6/19/39	5/21/39— 80 5/29/39— 62.5 6/10/39— 24	No shadow (5/27/39)
J. McM.	72	M	? Died 9/5/39 (no autopsy)	3/ 8/39— 45 3/10/39— 30 3/17/39— 37.5 3/28/39— 20	No shadow (3/17/39)
W. S.	56	M	? Died several months later	8/23/39—151 8/30/39—134 9/ 8/39—131 9/19/39—195	No shadow (8/25/39)

cystography should not be used in patients suffering from obstructive jaundice because fatal pancreatitis can sometimes follow such cases. In one of his own patients with slight jaundice a good shadow was obtained; the patient died ten days later from subacute yellow atrophy.

Foote and Carr,[6] in 1936, reported giving gallbladder dye (iodeikon) intravenously in fractionated dosage to 17 patients with jaundice without ill effect. As a result of this work and similar studies on dogs in which complete common duct obstruction had been produced, they stated that common duct (extrahepatic biliary) obstruction is demonstrated by the retention of the dye for forty-eight hours or longer in the extrahepatic biliary system. With intrahepatic obstruction the biliary system is not visualized after forty-eight hours, although a normal gallbladder shadow may be seen at twenty hours. They believe that we have here an aid in differentiating intrahepatic from extrahepatic biliary obstruction.

Jacobi,[9] in 1937, stated that visualization of the gallbladder in the presence of jaundice was obtained only in cases of toxic jaundice and agreed with Foley in the opinion that the absence of a shadow is of no value at all, but the presence of one confirms the existence of a non-obstructive jaundice. Visualization of the gallbladder with the oral administration of the dye was successful in 7 out of 8 of these cases in which it was attempted. The highest icterus index at which such visualization occurred was 66.

In 1937 Ottenberg,[14] changing his original position, stated that one is justified in attempting gallbladder visualization either by the oral or intravenous route in the presence of jaundice. His diagnostic criteria after this procedure are the same as those of Foote and Carr; namely, successful visualization followed by normal emptying points to hepatitis as the cause of jaundice; successful filling with failure to empty after the fatty meal points to obstructive jaundice; failure of visualization is of no differential or diagnostic aid. He still feels that there should be some selection of the cases in which cholecystography is attempted. Various tests are enumerated which are available as indices of the degree of liver damage. No cases are reported.

Behrens,[4] in 1940, stated that a gallbladder series is not often of much value in jaundice because, with the usual non-visualization, one cannot differentiate between an obstructive pathological condition or parenchymatous failure to excrete. With visualization and good emptying, obstruction is, of course, ruled out.

Buckstein,[3] in 1940, stated that his personal experience shows the procedure to be essentially innocuous and also may be of considerable aid in differential diagnosis. In jaundice he employs the following criteria: If a gallbladder fills and empties, jaundice is due to hepatitis; if there is a good shadow without emptying, the jaundice is of obstructive nature. No mention is made of the significance of no shadow. One case is reported in which the relatively faint shadow of an enlarged gallbladder was obtained in a patient with jaundice. There was no decrease in the size of the shadow after a fatty meal. Autopsy showed cancer of the pancreas with common duct obstruction.

In the past five years 50 unselected cases of jaundice have been submitted to cholecystography by the intensified oral method of Stewart and Illick.[17] There were no intravenous injections. No deleterious effects due to the procedure were noted.

Practically all of the cases had one or more determinations of the icterus index,[12] which is a quantitative estimation of the amount of bile pigment in the blood stream.

The largest group (15 cases) was that of common duct stone. Eleven showed "no shadow" of the gallbladder (opaque calculi were seen in two of the preliminary roentgenograms); faint gallbladder shadows were seen in the 4 remaining cases.

There were 3 cases of cholecystitis without cholelithiasis of which 2 had faint shadows; the other one no shadow.

The second largest group (11 cases) had no final diagnosis. A very faint shadow was obtained in only 1 case. The other 10 cases had no shadow.

The only other large group (10 cases) was that of toxic and infectious hepatitis (including so-called "catarrhal jaundice"). Good shadows were obtained in 7, faint shadows in 3 cases.

In the smaller groups, such as cancer of the pancreas (3 cases) a faint shadow appeared in 1, no shadow in the other 2 cases.

Similar findings occurred in the 3 cases of cancer of the common duct. Two cases of cancer of the liver were encountered, 1 case with and the other without a gallbladder shadow. No visualization of the gallbladder occurred in 1 case of cirrhosis of the liver and 1 case of duodenal ulcer associated with jaundice.

From a practical point of view the entire situation resolves itself into an attempt to distinguish between surgical and medical cases of jaundice. This was possible to a large extent in those 36 cases in which jaundice was decreasing. Since a patent common duct is here predicated, the same diagnostic criteria are used as in patients without jaundice. With increasing jaundice, however (the remaining 14 cases), diagnostic information is usually not obtained since apparently no difference exists in the degree of impairment of dye excretion whether due to back pressure (extrahepatic biliary obstruction) or to parenchymatous disease, either toxic, infectious, cirrhotic or neoplastic.

CONCLUSIONS

Cholecystography in jaundice, while apparently not harmful, is of little or no differential value when jaundice is increasing and when jaundice is decreasing may well be deferred.

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LAMINAGRAPHIC STUDIES OF THE AORTA*

THEIR ADVANTAGES AND LIMITATIONS

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ROENTGENOGRAPHIC examination of the thoracic aorta by the routine methods is all too frequently unsatisfactory. The difficulty lies largely in the fact that the aorta is in intimate contact with structures of similar density. Adequate visualization is further hampered by the overlying and obscuring shadows of the lungs and ribs. Three roentgenographic procedures are available for use in partially overcoming these difficulties. The first of these is the use of stereoscopic roentgenography in various positions. This spreads out the thoracic contents into a third dimension, but it does not eliminate the overlying shadows or provide a greater contrast in the density of the aorta over that of its neighboring structures. Since we are discussing roentgenographic methods, it is hardly necessary to mention here that roentgenoscopy is a very important part of every examination of the aorta.

The second procedure is the injection of a contrast medium into the blood stream and roentgenographing the aorta at the moment the contrast medium is in it. Without going into the details of cardiovascular opacification it requires experience and special equipment, and even then satisfactory visualization is not always obtained. The third method, and the one employed in this study, is body section roentgenography. The advantages of sectional roentgenography are: (1) it is a simple roentgenographic procedure that can be carried out by a technician; (2) the intravenous injection of a contrast medium is avoided; (3) the examination is not uncomfortable or hazardous for the patient, and (4) the shadows of overlying and obscuring structures are eliminated. Thus, on sectional roentgenograms, the aorta is seen with greater clarity and the observer's attention becomes automatically focused on it and the structures which lie in the same plane.

These advantages should not be inferred to mean that sectional roentgenography is superior to cardiovascular opacification. The latter is a feasible and excellent method of visualizing the aorta, and when successful, gives the most complete record of the aorta. Many times, however, the aorta can be sufficiently well demonstrated on the sectional roentgenograms without necessitating opacification of the vessel. It is for this reason that we are presenting these studies.

The laminagraph of Kieffer and Moore was used for all the sectional roentgenograms. It is well suited for the examination because by employing the spiral movement all the unwanted shadows are blurred out. This produces a sectional roentgenogram with a homogeneous black background undisturbed by incompletely "blurred out," distorted shadows of unwanted objects. It is this feature, in our opinion, that makes the laminagraph superior to other equipment for sectional roentgenography. The principles of sectional roentgenography[1,2,5] are now generally understood and will not be referred to at this time.

TECHNIQUE

The technical factors employed are the same as those used in previous studies of

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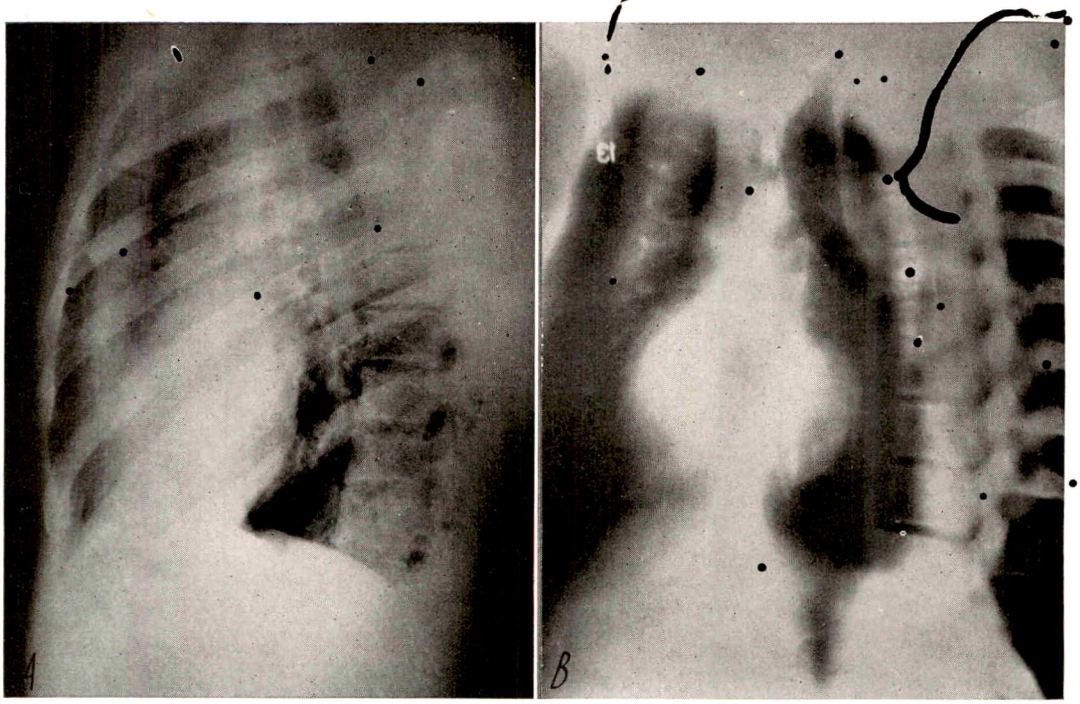


Fig. 1. Both routine and sectional roentgenographic examinations of the aorta in children and in young adults usually are not satisfactory. This is largely because the aorta is smaller in diameter, the walls are thinner, and the width of the aortic arch is narrower. *A*. On the conventional left anterior oblique chest roentgenogram of a healthy young intern, aged twenty-six, the borders of the aorta are hardly discernible. *B*. On the laminagram the aortic arch is better visualized, as are the great vessels leading to the neck. The descending aorta lacks sufficient density for adequate rendition.

the thoracic aneurysms[8] and of the pulmonary arteries;[9] namely, 60 milliamperes, 2½ to 3 seconds, 30 inch target-film distance, Lysholm grid, kilovoltage varied according to thickness, and with the spiral movement. The left anterior oblique position is used for visualizing the ascending portion of the arch of the aorta. The laminagram is usually made at a level opposite the right anterior axillary line. The left lateral position is used to record the descending portion of the arch of the aorta and the descending thoracic aorta. These laminagrams are made at levels 2 to 4 cm. lateral to the spinous processes. In the examination of each patient conventional roentgenograms are made first, and from a study of them it is possible to judge the positions and levels at which laminagrams would be most advantageous. In this way we avoid taking a needless number of sectional roentgenograms. This is the proper sequence in the use of sectional roentgenography in cardiovascular examinations, because if the structures in question are adequately demonstrated by routine methods, the use of body section roentgenography is not indicated. The following cases and illustrations will demonstrate these points and permit a discussion of various lesions of the aorta that are of clinical interest.

Arteriosclerosis of the Aorta

The aorta in a healthy young adult is difficult to visualize by either conventional or sectional roentgenography. The left anterior roentgenogram in Figure 1*A* was made of a young intern. The aorta in persons of this age is smaller, contains less calcium and fibrotic tissue, and therefore lacks the density of the aorta that is seen in older persons. Furthermore, the aortic arch is more acute and "bunched" together than is the wider arch of the mature adult. The

sectional roentgenogram in Figure 1*B* is an improvement. The great vessels entering the neck and the posterior portion of the aortic arch are fairly clear, but the descending aorta is scarcely differentiated from the surrounding tissues. Thus in children and in young adults, sectional roentgenography is not of much advantage.

It is in patients in the late forties, fifties, sixties, and beyond that age that sectional roentgenography is of value in examination of the aorta. In these patients arteriosclerotic changes have developed to various degrees. Microscopic sections through the wall of an arteriosclerotic aorta reveal that the intima is thickened and composed of dense, hyalinized connective tissue, in which acellular plaques and calcium deposits can be seen. These changes make the aortic wall thicker and of greater density as well as producing a lengthening of the aorta—all of which leads to better roentgenographic visualization. Examples of advanced arteriosclerosis are seen in Figures 2 and 3. In these illustrations note particularly the size (diameter) of the aortas and the superior clarity with which they are shown on the sectional roentgenograms. Even in these advanced cases of arteriosclerosis the diameter of the aorta is only slightly enlarged over that of individuals in the late forties and early fifties, but it is greatly lengthened, and there is a marked spread in the distance between the ascending and descending aortas.

DISSECTING ANEURYSMS OF THE THORACIC AORTA

An important but seldom recognized complication of advanced arteriosclerosis of the aorta is the development of a dissecting aneurysm. An associated hypertension is the usual accompaniment in the formation of this complication. Shennan[10] in 1934 reviewed the world literature and collected 300 cases, but only 6 of these had been

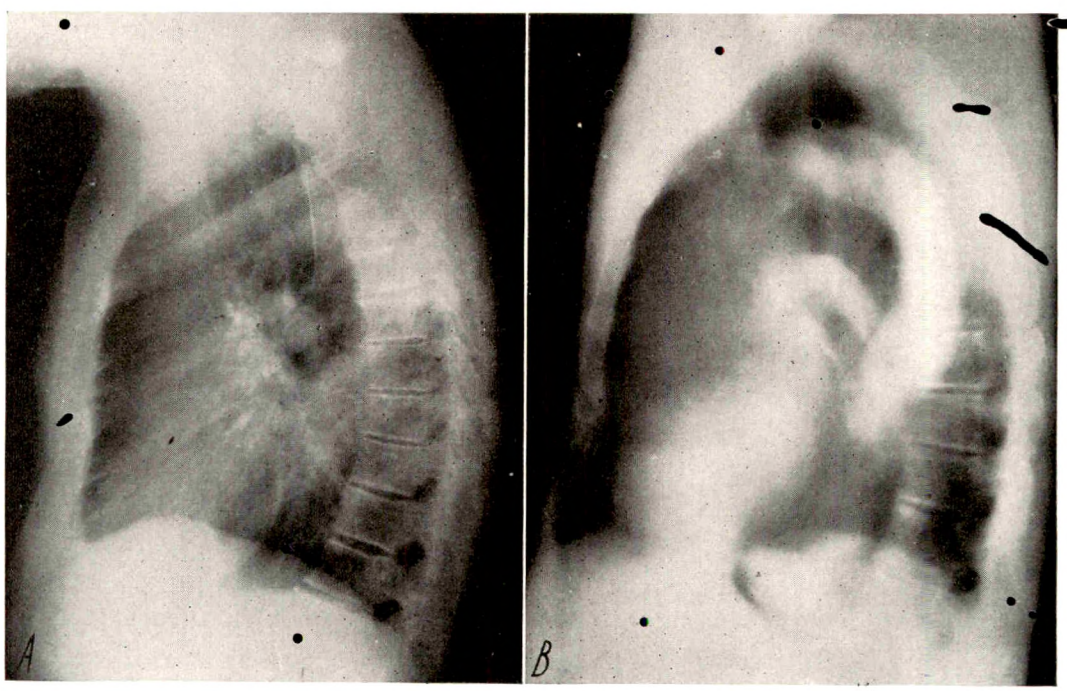


FIG. 2. Illustrates moderate arteriosclerosis of the aorta in a man, aged sixty. The aorta is lengthened, but only slightly enlarged in diameter. Blood pressure 140/85. Serology negative. *A*. The conventional left lateral view of the chest provides an unusually clear visualization of the aorta. *B*. The sectional roentgenogram affords even better definition of the aorta. Compare the aortic size in Figures 2 and 3 with those in Figures 4, 5 and 6.

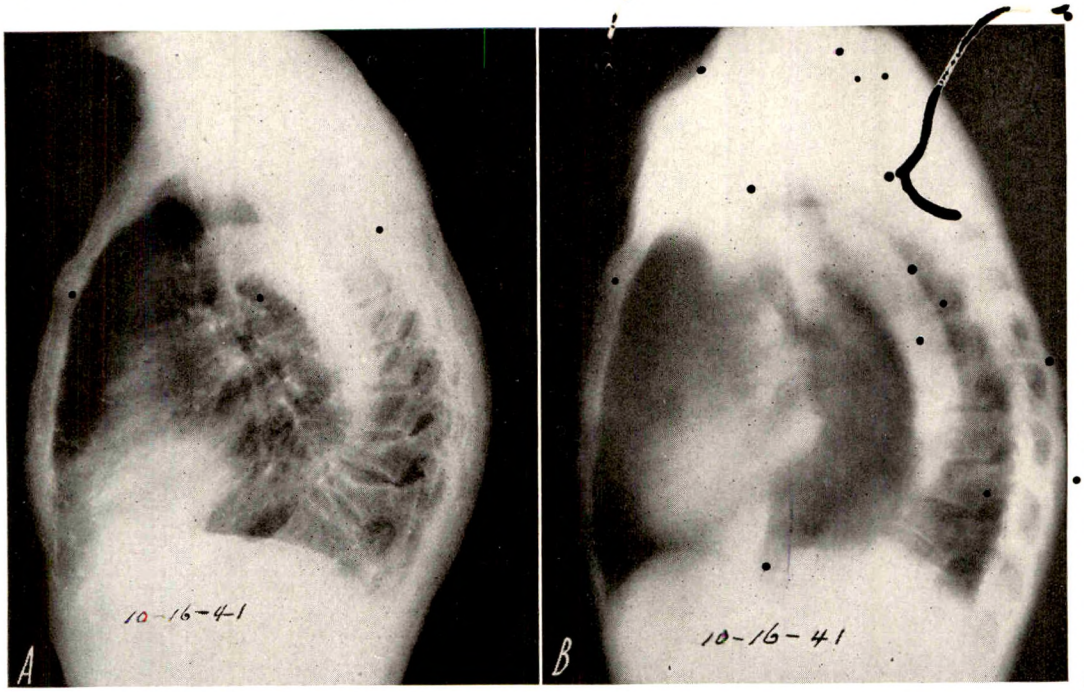


FIG. 3. In advanced arteriosclerosis, as illustrated in these roentgenograms of a woman, aged seventy-nine, there is marked lengthening and increased tortuosity of the aorta without appreciable increase in the size of the diameter of the aorta. *A*. Conventional left lateral roentgenogram of the chest. *B*. Left lateral sectional roentgenogram. Note the shadows of the great vessels leaving the aorta to enter the neck and that of the inferior vena cava.

diagnosed before death. Since then excellent clinical studies have been published by McGeachy and Paullin,[4] Leary and Weiss,[3] Rottino,[6] Ruffin, Castleman and White,[7] and a correct antemortem diagnosis is no longer a rarity. Part of the difficulty of diagnosis lay with the internists, who were not familiar with the symptoms of this complication, and part with the roentgenologists, because they were unable to demonstrate the aortic change adequately. The attempt to establish this diagnosis in the following 3 cases was the stimulus for this paper.

CASE 1. The patient was a physician, aged sixty-three, who first entered the hospital in September, 1940, seriously ill with a pneumonia. The roentgenogram in Figure 4*A* was made during his convalescence. The aorta is of average size and shape for a man of his age with a hypersthenic habitus. Six months later while on a business trip he was suddenly stricken with a severe pain in the middle of the thoracic back. The pain was constant, radiated to the scapulae and required large doses of morphine for relief. Coronary thrombosis was suspected but the electrocardiogram was negative. After three days of hospitalization he returned home. On the next day he again experienced this severe pain and was hospitalized and required morphinization to control the pain. The pulse, temperature, respiration, heart sounds and laboratory examinations were all negative, including another electrocardiogram. The blood pressure was 170/102 and was equal in all extremities. A roentgenogram of the chest at this time showed a definite widening of the aortic shadow (Fig. 4*B*). A widening of this degree occurring in a short period of time together with a severe constant pain in the mid-thoracic back is the salient sign and symptom of a dissecting aneurysm. In the lateral view (Fig. 4*C*), the aortic arch appears widened, but it is not sufficiently clear to establish the diagnosis. The lateral sectional roentgenogram (Fig. 4*D*) shows the very marked increase in the width of the aortic arch and the proximal descending thoracic aorta with sufficient detail to establish the diagnosis

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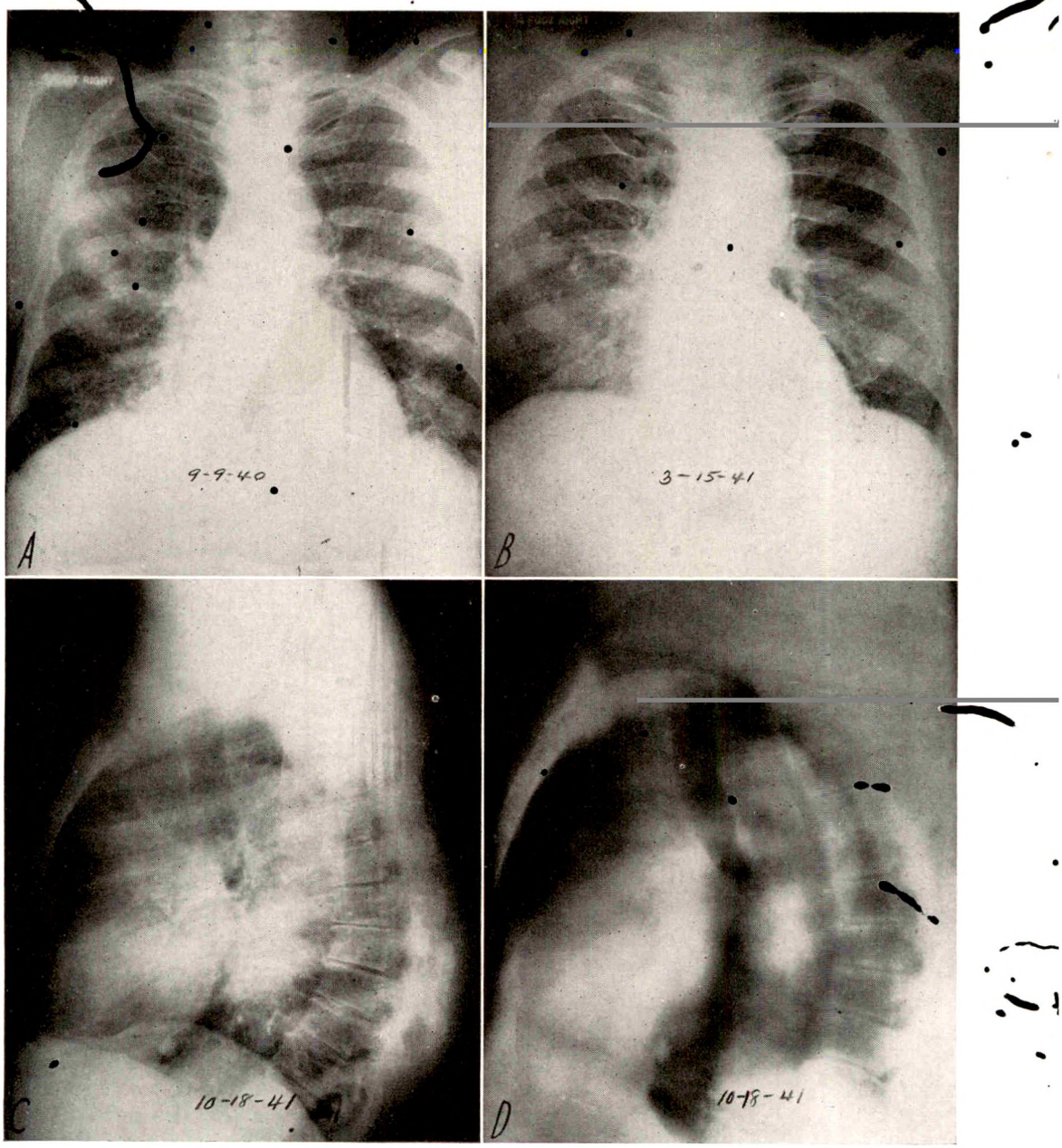


FIG. 4. Case 1. Dissecting aneurysm of the arch and descending portion of the thoracic aorta in a physician, aged sixty. *A*. Routine posteroanterior chest roentgenogram made on September 19, 1940, during his convalescence from a serious attack of bilateral pneumonia. The aortic shadow is slightly widened, but no more than is usually seen in a man of this age with a hypertension of 170/102. *B*. Posteroanterior chest roentgenogram made on September 15, 1941, at the time the patient experienced a severe boring pain in the mid-thoracic back. This degree of widening of the aortic shadow occurring in a short period of time should always suggest the possibility of a dissecting aneurysm. *C*. Left lateral roentgenogram of the chest confirms a definite enlargement of the aortic arch and proximal descending aorta. *D*. Left lateral laminagram reveals more clearly, and therefore with greater certainty, the marked enlargement of the arch and descending aorta due to the dissecting aneurysm. The trachea and bifurcation are in the same plane and are clearly shown.

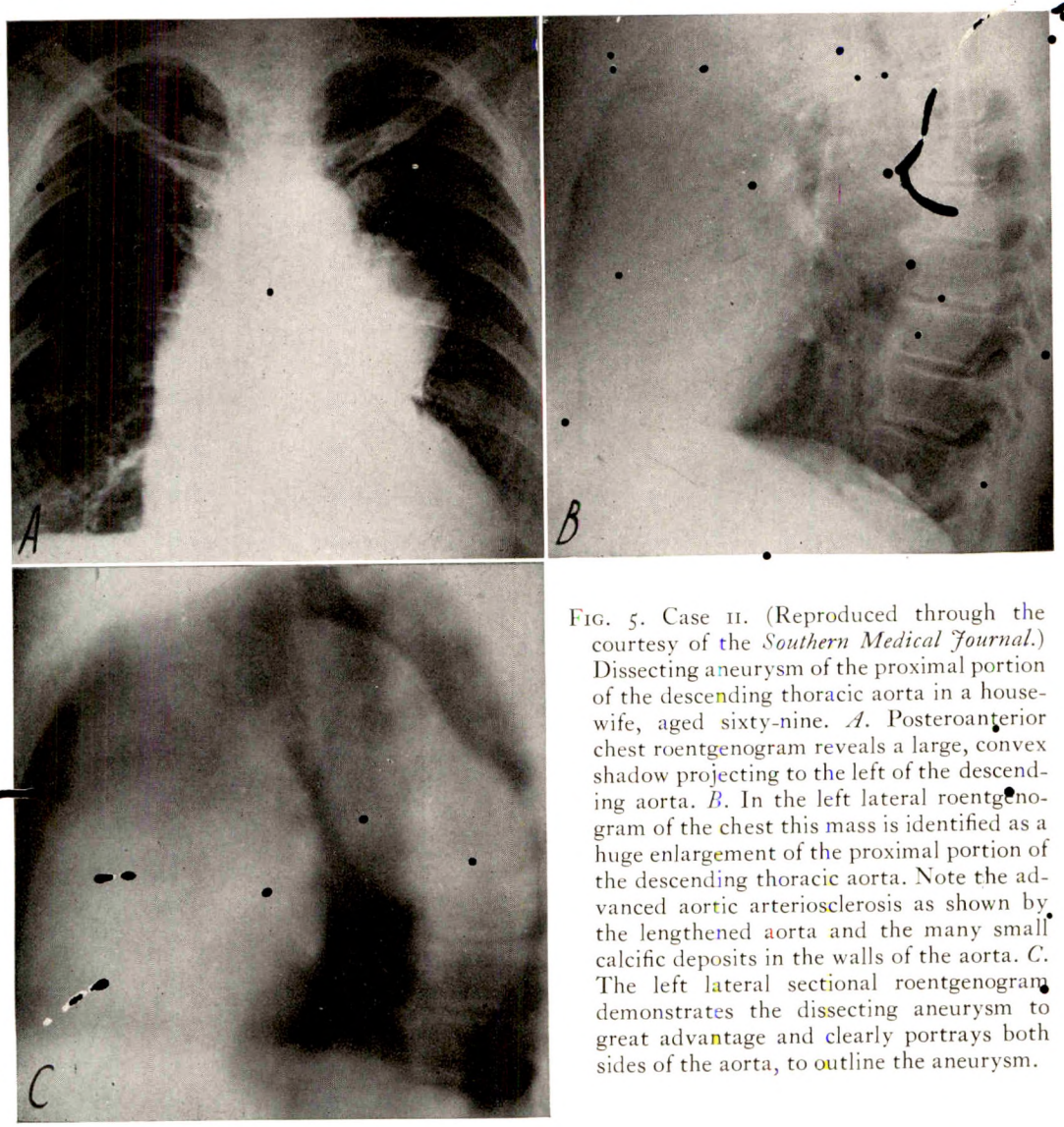


FIG. 5. Case II. (Reproduced through the courtesy of the *Southern Medical Journal.*) Dissecting aneurysm of the proximal portion of the descending thoracic aorta in a housewife, aged sixty-nine. *A.* Posteroanterior chest roentgenogram reveals a large, convex shadow projecting to the left of the descending aorta. *B.* In the left lateral roentgenogram of the chest this mass is identified as a huge enlargement of the proximal portion of the descending thoracic aorta. Note the advanced aortic arteriosclerosis as shown by the lengthened aorta and the many small calcific deposits in the walls of the aorta. *C.* The left lateral sectional roentgenogram demonstrates the dissecting aneurysm to great advantage and clearly portrays both sides of the aorta, to outline the aneurysm.

of a dissecting aneurysm with reasonable certainty. His pain gradually subsided in the following three months and he has been able to return to his work.

CASE II. The second case was a housewife, aged sixty-nine, who was admitted to the hospital on April 8, 1940, during an attack of influenza. After the acute symptoms subsided she was carefully examined because of a history of a persistent cough for the past year accompanied by a dull pain in the left chest and back. The onset of these symptoms was sudden. Since then she had never felt well and her condition had been steadily growing worse. On physical examination the blood pressure was 184/108. A diastolic murmur was heard over the region of the aortic valve. Laboratory reports were negative, including the serology. The electrocardiogram revealed evidence of a left axis deviation. The first roentgenogram of the chest (Fig. 5*A*) was made the day she entered the hospital. A large, convex shadow was found in the region of the descending thoracic aorta. The appearance of this suggested the possibility of a mediastinal tumor. The heart was moderately enlarged to the left. In the left lateral roentgenogram (Fig. 5*B*) this shadow is seen to be the result of a huge enlargement of the aorta, and without doubt is a dissecting aneurysm. On the sectional roentgenogram (Fig. 5*C*) the dissecting aneurysm is beautifully pictured, and we doubt if opacification could

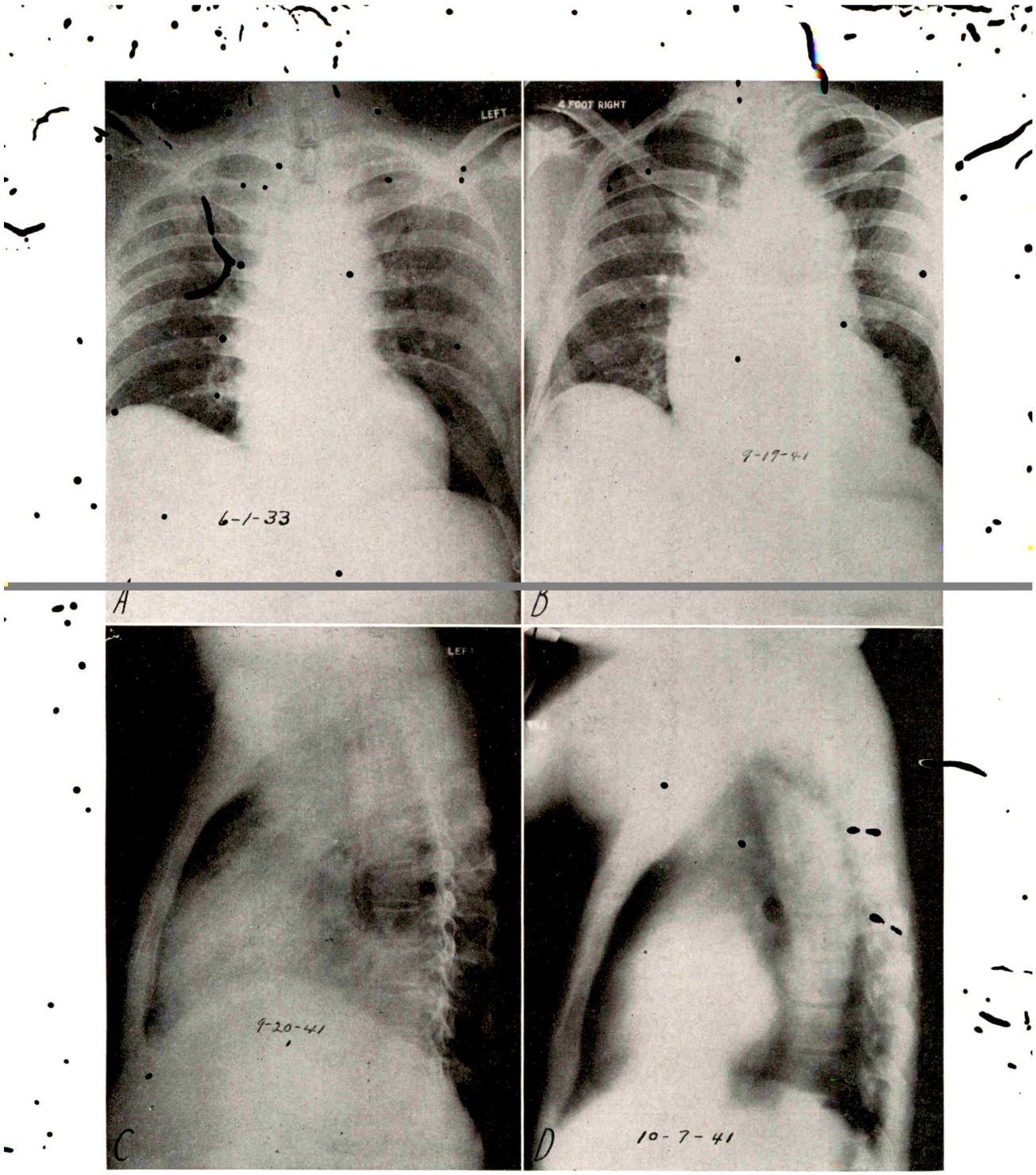


FIG. 6. Case III. Dissecting aneurysm involving the ascending portion and arch of the aorta in a male, aged fifty-two, with a hypertension of 160/90. *A*. The posteroanterior roentgenogram of the chest was made on June 1, 1933, at the time he had a severe attack of precordial pain that was considered to be a coronary thrombosis. However, in the light of subsequent developments, it seems possible that this episode was the initial break in the intima with beginning dissection. This view is compatible with the degree of widening of the aortic shadow present at this time. *B*. This chest roentgenogram was made September 19, 1941, at the time of his second attack of severe pain in the chest and back. The great enlargement of the aorta at once suggests an aneurysm. *C*. The left lateral view is difficult and unsatisfactory to interpret, as the detail is poor, due to the large size of the patient. *D*. On the left lateral laminagram the huge enlargement of the aortic arch is well demonstrated, and diagnosis of a dissecting aneurysm is as certain as it is possible to determine without autopsy confirmation.

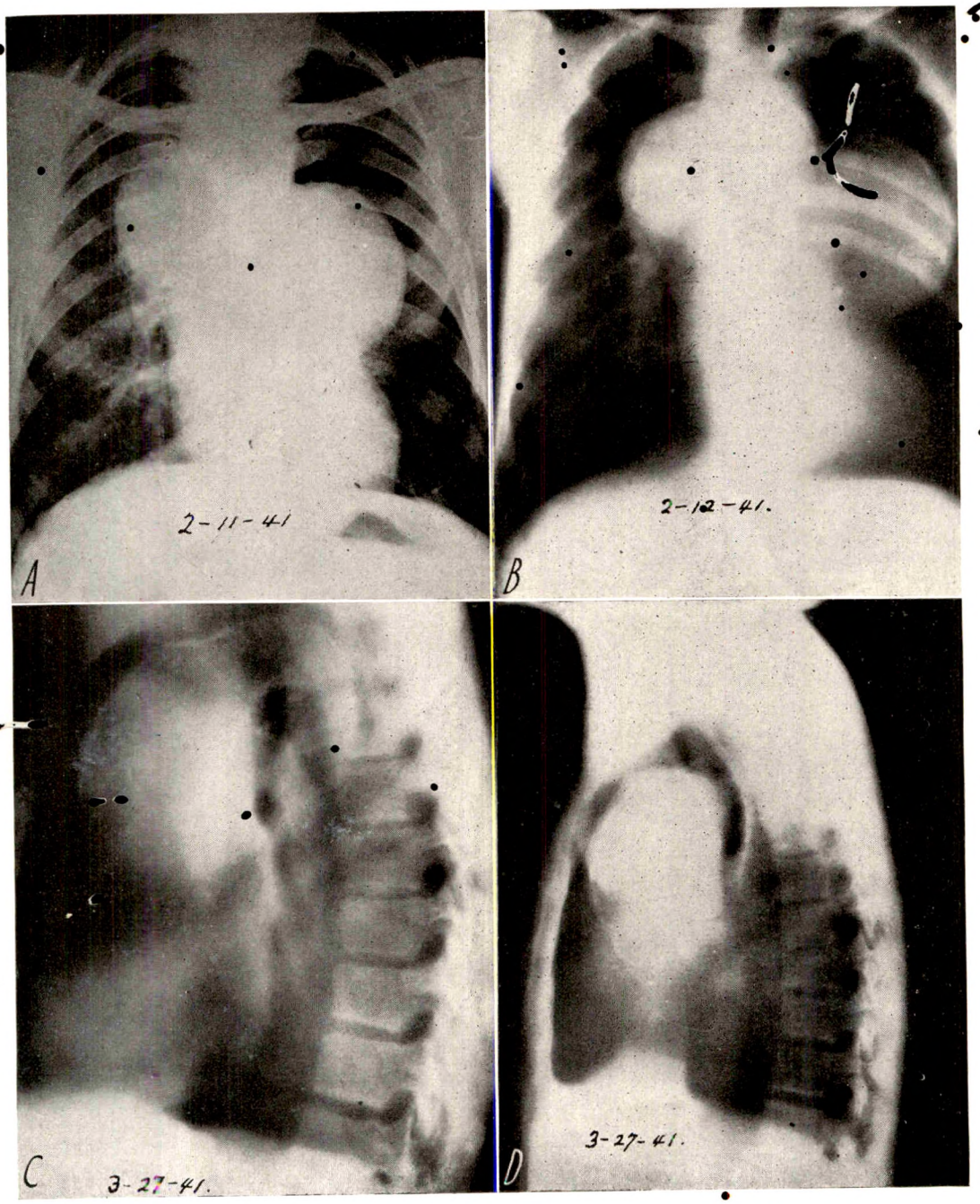


FIG. 7. Case IV. A double, sacculated aneurysm in a male, aged sixty-three. *A*. On the posteroanterior chest roentgenogram it is impossible to identify the sites of origin of the aneurysms and what segments of the aorta they involve. *B*. On the posteroanterior sectional roentgenogram the deposits of calcium in the walls of the ascending aorta and in the aneurysms are sufficiently well demonstrated to outline these structures. The mouths to both aneurysms are visible, and, since they and the ascending aorta are both in the same plane, the aneurysms both arise from this portion of the aorta. *C* and *D*. In the conventional left lateral roentgenogram both aneurysms overlie each other, and the details of each are obscured. Sectional roentgenography makes it possible to take a section through each aneurysm separately. *C* is through the large aneurysms on the left, and *D* is through the aneurysm on the right. Note the opening of the aneurysm into the aorta.

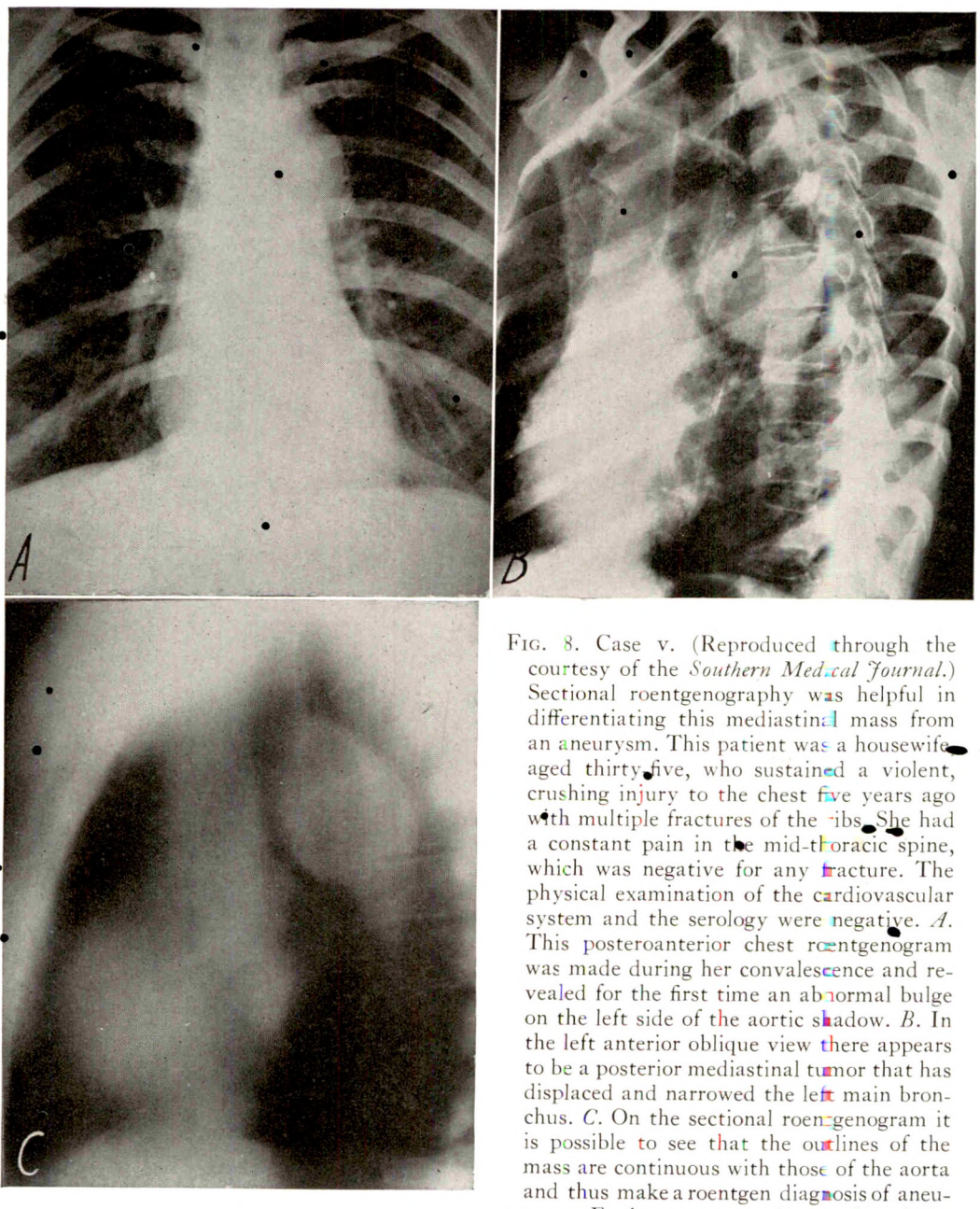


FIG. 8. Case v. (Reproduced through the courtesy of the *Southern Medical Journal.*) Sectional roentgenography was helpful in differentiating this mediastinal mass from an aneurysm. This patient was a housewife aged thirty-five, who sustained a violent, crushing injury to the chest five years ago with multiple fractures of the ribs. She had a constant pain in the mid-thoracic spine, which was negative for any fracture. The physical examination of the cardiovascular system and the serology were negative. *A.* This posteroanterior chest roentgenogram was made during her convalescence and revealed for the first time an abnormal bulge on the left side of the aortic shadow. *B.* In the left anterior oblique view there appears to be a posterior mediastinal tumor that has displaced and narrowed the left main bronchus. *C.* On the sectional roentgenogram it is possible to see that the outlines of the mass are continuous with those of the aorta and thus make a roentgen diagnosis of aneurysm. Exploratory operation confirmed the roentgen diagnosis.

demonstrate it to better advantage. We have not been able to trace this patient since she left the hospital.

CASE III. The third case was a male, aged fifty-two, store manager. His first chest roentgenogram was made in June, 1933, when he entered the hospital with hypertension and symptoms of a coronary thrombosis (Fig. 6*A*). Even at that time he had a definite widening of the aortic shadow and it is possible that this incident was the first break in the intima with the beginning dissection that ultimately developed into an aneurysm. In 1940 he had a cerebral accident and developed an aphasia from which he fully recovered. On September 15, 1941, eight years after the first chest roent-

genogram, he was stricken with a severe pain in the lower thoracic and upper lumbar spine. The pain radiated to the left side of the abdomen. He was slightly dyspneic, pale, anxious and covered with a cold sweat. The blood pressure was 160/90. The electrocardiogram was not even suggestive of a recent cardiac infarction. The laboratory reports, including the serology, were all negative. A chest roentgenogram was made, and the great increase in the aortic width at once suggested the diagnosis of a dissecting aneurysm (Fig. 6*B*). The conventional lateral roentgenogram (Fig. 6*C*) is not particularly helpful. This patient is a big man, and the outlines of the aorta are indistinct. On the left lateral sectional roentgenogram (Fig. 6*D*) the extreme enlargement of the aortic arch and descending portion of the aorta is very evident and the diagnosis of a dissecting aneurysm is as certain as is possible without an autopsy. This patient is still able to be up and around.

These 3 cases bring out the fact that the roentgenographic examination is a most important factor in the diagnosis of dissecting thoracic aneurysms and that sectional roentgenography is a definite help.

Sectional roentgenography can be employed with equal effectiveness in visualizing sacculated thoracic aortic aneurysms. The following case of a double aneurysm illustrates very well the advantage offered by sectional roentgenography.

Case iv. The patient was a male, aged sixty-three. He had complained of dyspnea and palpitation for the past ten months. There was a marked systolic murmur over the aortic and pulmonic areas. The Wassermann reaction was 4 plus and the blood pressure 140/72. In the posteroanterior conventional roentgenogram (Fig. 7*A*) it is impossible to determine the division or divisions of the aorta that are involved by the aneurysms. A laminagram (Fig. 7*B*) at the level of the ascending aorta brings out the fact that both aneurysms arise from the ascending aorta and are directly opposite each other. An almost continuous deposit of calcium forms the outlines of these structures. By tracing the left border of the aorta one can see the opening of the larger aneurysm, the border of which is easily followed back to the mouth and across to the arch of the aorta. The mouth of the smaller aneurysm is not as prominent but can be made out. Since both aneurysms lie at the same depth and opposite each other, it is obvious that on the conventional lateral roentgenogram their shadows will be superimposed obscuring the details of each. By sectional roentgenography, however, a separate lateral film can be made of each aneurysm as shown in Figures 7*C* and 7*D*. The sectional examination is not necessary to establish the diagnosis in this case, but it does demonstrate another way in which sectional roentgenography can be used and which may be of value in the diagnosis of other cases.

Differential Diagnosis between Aneurysms and Mediastinal Tumors

Sectional roentgenography occasionally has been helpful in distinguishing between aneurysms and mediastinal tumors. It is frequently possible to visualize a questionable mass with greater clarity on the sectional roentgenogram, and in this way determine if its outlines are continuous with those of the aorta. If so, then it is in all probability an aneurysm as both are at the same level. The following case is an excellent example of this point, and even though it has been previously published,[8] it seems worth repeating.

Case v. The patient, a female, aged thirty-five, received a severe crushing injury to the chest in an automobile accident five years ago. At that time she complained of a severe pain in the mid-thoracic spine. Roentgenograms of the spine, the physical examination, and the serology were negative. A chest roentgenogram revealed an abnormal bulge in the region of the distal part of the aortic arch (Fig. 8*A*). In the left anterior oblique view it appears to be a rounded mass causing pressure on the left main bronchus (Fig. 8*B*). The overlying and obscuring shadows are eliminated in the left anterior oblique laminagram (Fig. 8*C*), and it is possible to see that the outlines of the mass are continuous with those of the aorta anteriorly and posteriorly, which identifies it as an aneurysm, and we believe it to be a traumatic aneurysm.

Conclusion

In conclusion, the advantages and limitations of sectional roentgenography when applied to the study of aortic arteriosclerosis, dissecting aneurysms and saccular aneu-

rysms of the thoracic aorta can be summarized as follows:

1. Body section roentgenography is a new method of examining the thoracic aorta that is of definite value in the diagnosis of arteriosclerosis, of dissecting aneurysms and of sacculated aneurysms. The examination is simple, can be done by a technician, and is not uncomfortable or hazardous to the patient.

2. The laminagraph of Kieffer and Moore is well suited for taking sectional roentgenograms of the aorta because the unwanted shadows are dispersed in every direction to provide a uniform background.

3. The conventional, routine roentgenographic examination should always be made first, as a study of these roentgenograms will aid in determining the positions and levels at which to make laminagrams.

4. Sectional roentgenography of the aorta should not be used as a routine procedure but as a special examination in selected cases. It is least applicable in children and young adults and most effective in older persons with arteriosclerosis.

5. Emphasis is placed on the importance of recognizing the symptoms of dissecting aneurysms of the thoracic aorta so that suspected patients would be submitted to a complete roentgen study including sectional roentgenography.

6. Adequate roentgenographic visualization of thoracic dissecting aneurysms is the cardinal factor in establishing this fairly common but seldom recognized diagnosis. Sectional roentgenograms, in our experience, provide the most satisfactory method of demonstrating these aneurysms.

7. Saccular aneurysms can be visualized to the same advantage and can occasionally be distinguished from mediastinal tumors when a continuous outline is demonstrated between the mass and the aorta.

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VOL. 51, NO. 1

PRACTICAL CARDIOKYMOGRAPHY

ITS SIGNIFICANCE IN EVALUATING CARDIAC FUNCTION

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CAPTAIN, MEDICAL CORPS, A.U.S.

THE theoretical basis of cardiokymography, as well as the changes which are noted to accompany structural cardiac abnormalities, has been discussed in detail in recent medical literature. It is our purpose to direct attention to the contributions roentgen kymography may offer in the evaluation of cardiac functional capacity in the presence of heart disease or extracardiac disorders.

The conclusions represent a study of a series of 1,000 cardiokymograms during 1940 and 1941. The indications for these studies were structural and functional cardiac abnormalities, as well as diagnostic aid in differentiating various juxtacardiac masses.

The apparatus employed was a conventional type; namely, a multiple slit kymograph with a grid width of 12 mm. and a slit width of 0.4 mm. The target film distance was 30 inches, with an exposure time of 1.5 seconds. Posteroanterior exposures were routinely employed.

An introductory discussion of the normal kymographic silhouette will be appropriate (Fig. 1).

In the analysis of cardiograms the appearance of the left cardiac contour receives the primary and precise evaluation; the pulsations on the right, however, while they are also of fundamental importance, especially in a consideration of variations in cardiac rhythm and function, cannot always be analyzed with the same precision. The reason for this will be discussed in the text.

Along the lower left border may be seen the characteristic pulsations of the left ventricle. The diastolic period is biphasic with an initial rapid filling followed by a stage of slower filling of variable character; when this diastolic pulsation is flattened, it is described as a plateau. In many cases

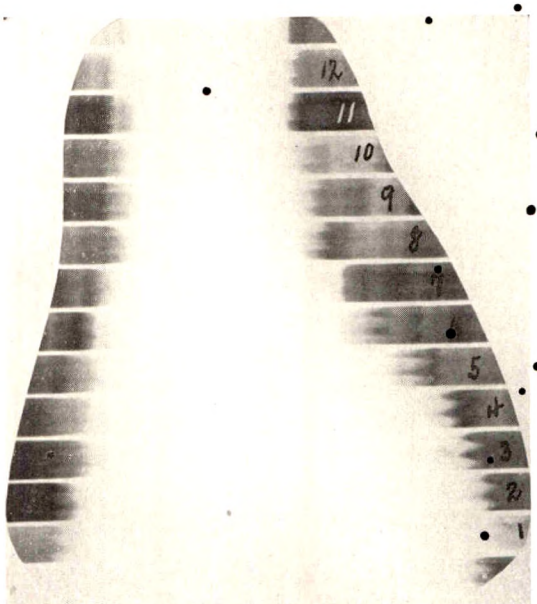
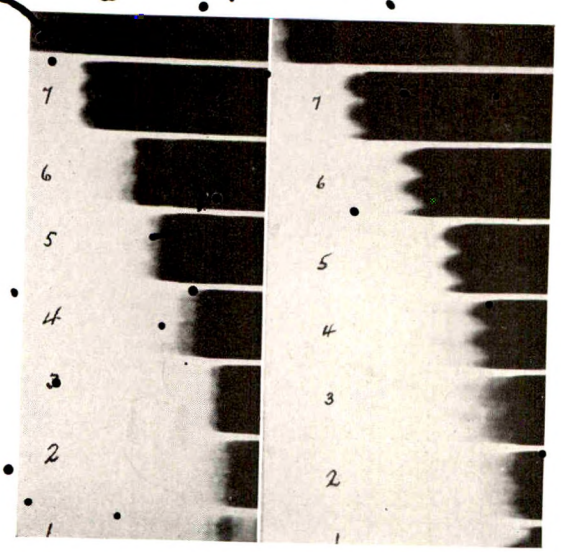


FIG. 1. Normal male, aged twenty-seven. Frames 3 to 7 on right show right auricular and ventricular pulsations; frames 8 to 12 show ascending aorta and vena cava. Frames 3 to 6 on left show left ventricular pulsations; frame 7 shows left auricular and ventricular pulsations; frames 8 to 12 depict the aorta.

it is replaced by a peak due to a shortened filling phase. This is followed by a sharp inward impulse representing a ventricular systole.

The aortic pulsations are in opposite phase to that of the left ventricle; namely, the outward thrusts represent systole, and the troughs obviously represent the diastolic phases. These expansions and recoils

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A B

FIG. 2. *A*. Male, aged fifty-two. Hypertensive; arteriosclerotic; pulse 140+. Roentgen kymogram of left ventricle reveals rapid and low amplitude pulsations. *B*, roentgen kymogram of same patient one week later after digitalization shows normal amplitude in frames 4 to 7. Pulsations almost obscured in frames 2 and 3 due to myocardial infarction.

of the thoracic aorta are evident in Figure B. There is a perceptible lag between ventricular pulsations and aortic impulses. Since each frame represents the same time interval, the time relationship between the various chambers may be compared by measuring the position of the impulses of the corresponding frame. The pulmonary artery pulsations, similar in character to the aorta, though not as vigorous or sharply peaked, may be visualized in the upper frames. The pulsations of the left auricular appendage are characteristically barely perceptible.

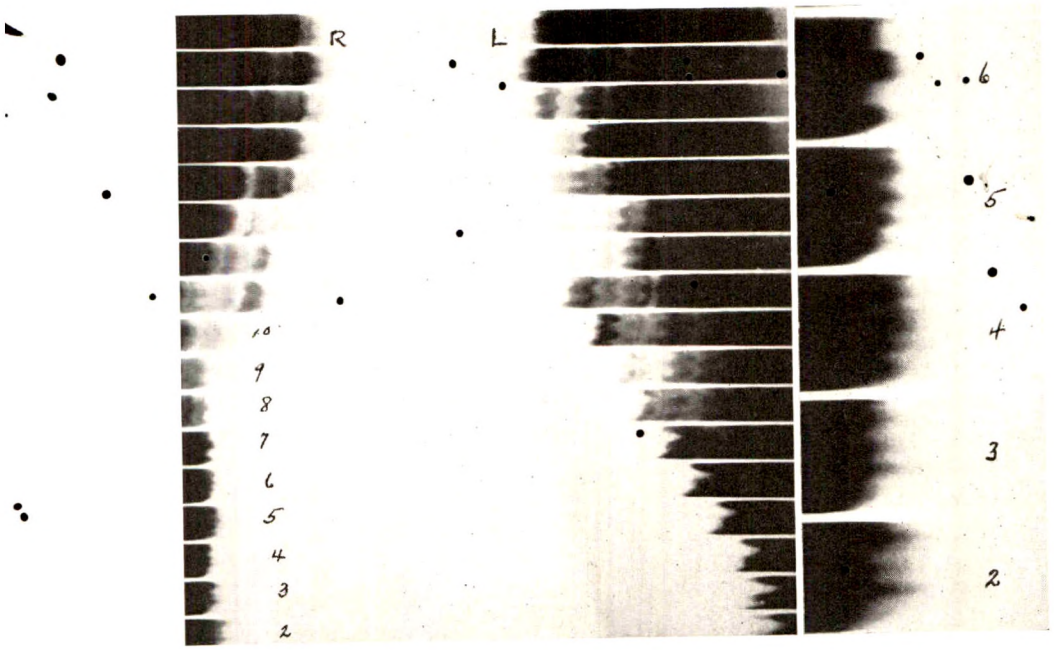
Progressing from below upward along the right border, the right ventricular contractions may be seen; they are similar in character to those of the left ventricle. In the region of the right auricle, two types of pulsations are evident. There is a transmitted wave of a ventricular character which is dominant, as well as an independent auricular impulse. The latter may be seen as a low amplitude rhythmic wave. It is often difficult to determine the character of these waves in the presence of marked pulmonary congestion. Along the upper portion of the right border, the contours represent the combined effect of the superior vena cava as well as the pulsations of the aorta.

Besides evaluating the character and the time relationship of the various pulsations, it is important to consider their amplitude. In a characteristic ventricular wave this may be done by measuring the distance between the peak and the trough. While it is true that such measurements are related mainly to the horizontal component of motion, they are nevertheless of significance. With the above described technique, the amplitude of normal ventricular curves varies from 4 to 8 mm.; for such measurements, frames 3, 4, 5 and 6 on the left are most reliable. The lower two frames, while they show pulsations, are not as valid guides; their amplitude is usually depressed. This is probably due to the dominance of the vertical and rotary components over the horizontal. The pulsations in these lower frames may be completely obliterated by minimal pericardial effusion. The presence or absence of such a complication should be determined in analyzing the amplitude of corresponding frames of both sides.

Cardiac physiologic and functional capacity may be indicated kymographically by a consideration of rate and rhythm as well as the intensity of the pulsatile phenomena.

Tachycardia is evidenced by an increase in the number of pulsations in each frame. In sinus tachycardia, the auricles and ventricles exhibit equal, regular and frequent pulsations (Fig. 2, *A*).

In the presence of a totally irregular ventricular rhythm, there is noted varying amplitudes of the left ventricular pulsations in a single frame. This is apparently related to a shortening and, hence, inequality of the diastolic phases. In auricular fibrillation, the ventricles exhibit these phenomena, usually at a rate of between 60 and

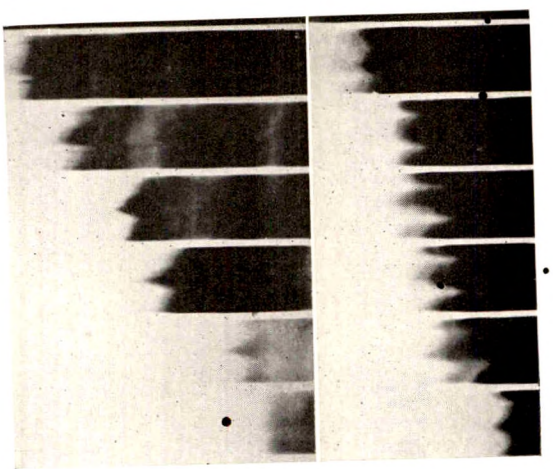


A B

Fig. 3. *A*. Male, aged fifty-seven. Pulse 88, irregular in rate; presystolic murmurs at apex; blood pressure 148/90; electrocardiogram, auricular fibrillation. Roentgen kymogram shows on left pulsations of irregular amplitude in frames of ventricle. On right, frames 2 to 10 show the pulsations of right ventricle and a fibrillating right auricle. *B*, magnified view of frames 2 to 6 of right side.

100 per minute, while the auricular waves along the right cardiac border appear as an irregular, poorly defined, serrated pattern (Fig. 3, *A* and *B*). Frequent ventricular extrasystoles may give a similar ventricular pattern; the extrasystole appears as a wave of reduced amplitude. The auricular pulsations on the right, however, are well defined.

Cardiac functional capacity may, too, be properly indicated. Typically, there is noted in cardiac failure a reduction in ventricular amplitude. From a normal level between 4 and 8 mm., the amplitude declines below 4 mm. It is true that this does not truly represent the vertical or rotary phenomena. However, in the presence of cardiac hypertrophy or dilatation, the amplitude of pulsation may be reduced without any gross reduction in cardiac output. Nevertheless, there is still found a close correlation between cardiac failure and the marked reduction in amplitude in the ventricular pulsations. Here, there is often noted a similar variation in aortic amplitude.



A B

Fig. 4. *A*. Male, aged fifty-eight. Clinical diagnosis: myxedema. Pulse 68; basal metabolic rate −28 per cent. *B*, same patient after thyroid therapy. Pulse 104; basal metabolic rate −6 per cent. Compare the amplitude and rate of left ventricular pulsations in the two illustrations.

In the presence of cardiac failure, the administration of digitalis produces recognizable kymographic changes. There is noted a gradual reduction in cardiac rate. The ventricular pulsations increase in amplitude until they may reach a normal or higher level, while the diastolic phase may become prolonged (Fig. 2, *A* and *B*). These changes have been found to occur consistently in a series of cases studied kymographically before and after digitalization. Following the treatment of beriberi and myxedema, similar kymographic changes are observed (Fig. 4, *A* and *B*).

In conclusion, it is felt that cardiokymography is of value in considering cardiac physiologic and functional capacity. It is further evident that though the wave recorded in a single frame is usually the pattern of a single muscular unit, the kymograph does not record the vertical and rotary phenomena in any precise fashion. Moreover, cardiac size and tone is an influencing factor upon the pulsatile cardiac amplitude.

Nevertheless, it is felt that if one is conscious of the limitations referred to above, cardiokymography can contribute much information of clinical value.

SUMMARY

1. Attention is directed to the value of roentgen kymographic study as an index of cardiac rhythm and functional capacity.

2. It is a simple and precise record of heart function in cardiac or extracardiac disorders.

3. Its value in prognosticating the response to therapeusis is emphasized.

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ANGIOCARDIOGRAPHIC ANALYSIS OF THE CARDIAC CONFIGURATION IN RHEUMATIC MITRAL DISEASE

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THE cardiac configuration in rheumatic mitral stenosis ordinarily is sufficiently distinctive in the conventional roentgenogram to suggest the diagnosis of the lesion. However, analysis of the cardiac contours, especially the left, has been the subject of considerable discussion. In the past, studies have been based on postmortem material. Correlation with the gross anatomical topography has been made ordinarily, although some investigators have made roentgen studies after chamber opacification in the intact cadaver.

As a result of these studies, there is a difference of opinion regarding the analysis of the segment of the left cardiac contour situated between the aortic knob and the left ventricle. Part of the confusion is due undoubtedly to the lack of uniformity in nomenclature. In the present discussion the term "pulmonic conus" is used to designate that portion of the right ventricle immediately below the pulmonary valve, i. e., the outflow tract. Although this is the accepted standard terminology, the term has been applied to the base of the pulmonary artery, which we regard as the pulmonary sinus. Furthermore, atrium and auricle will be used for those structures which sometimes are referred to as auricle and auricular appendage respectively.

In the mitral configuration, the middle segment of the left cardiac contour may be divided into two arcs. The upper arc, immediately caudad to the aortic knob, represents the main pulmonary artery or its left branch. All authors agree with this interpretation. Which structure forms the lower arc, however, has been a controversial issue. Groedel[3] concluded, on the basis of postmortem studies, that, in mitral stenosis, this arc is formed by the left atrium solely. Assmann[1] suggested, on the basis of similar studies, that the left auricle rather than the atrium was the component. Both agreed that only in advanced cases does the pulmonic conus contribute to the formation of this arc. Holzman,[4] Zdansky,[14] Uhlenbruck,[13] and Teschendorf[12] concur with this interpretation.

An opposing interpretation is advanced by Roesler,[8] Laubry *et al.*[5] and Dressler.[2] These authors, also on the basis of postmortem studies, conclude that in mitral stenosis the pulmonic conus forms the lower arc of the mid left cardiac contour. They emphasize that, with rare exceptions, the left atrium and auricle do not appear on the left border. This concept is accepted by some American cardiologists and roentgenologists[3] who attribute any prominence of the middle left segment to enlargement of the pulmonic conus. Indeed this was approximately the interpretation accepted by one of the present authors.[9] On the other hand, Robb and Steinberg,[7] who first employed angiocardiography in the study of rheumatic heart disease, disagreed with this interpretation.

Since postmortem studies led to such divergent conclusions, it was evident that studies made during life would be desirable. Angiocardiography was therefore employed in 28 cases, using the multiple roentgen exposure technique.[10,11] Diodrast 70 per cent (Winthrop) was injected rapidly intravenously as described by Robb and Steinberg.[7] The examinations were preceded in each case by complete clinical, electrocardiographic and phonocardiographic, as well as conventional roentgen studies. Our conclusions are based upon a correlation of these findings.

The cases were selected both from the

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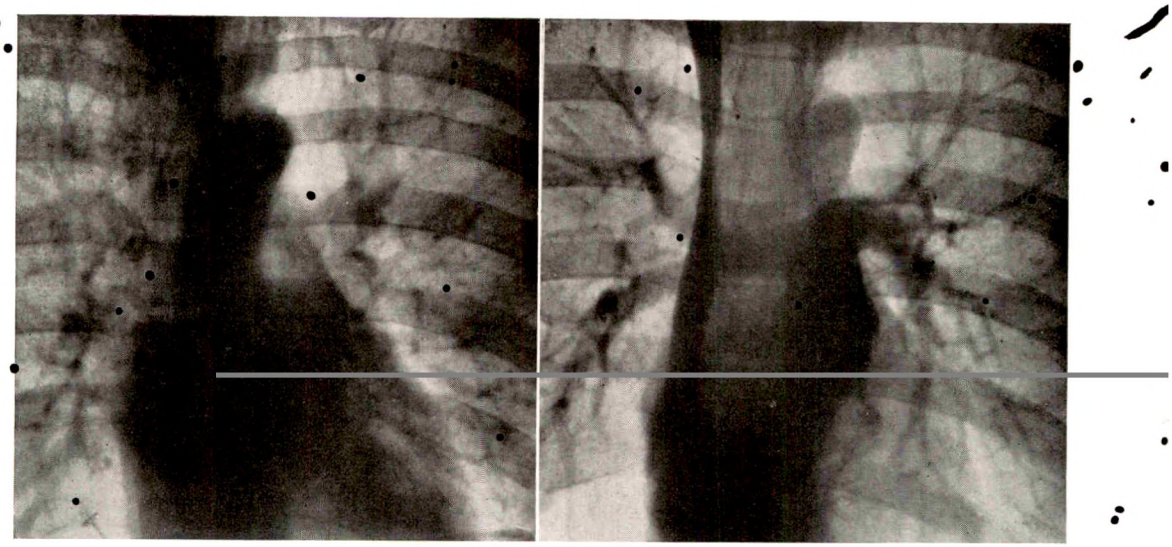


FIG. 1. Case 10. Mitral stenosis. Angiocardiography. Roentgenogram on the left shows opacification of the left auricle, left ventricle and aorta. Roentgenogram on the right shows right auricle, right ventricle and pulmonary arteries.

in-patient and out-patient departments so that the group might include all stages of mitral disease. The cases therefore include examples of mitral insufficiency and stenosis, with and without involvement of other valves and with varying degrees of atrial enlargement.

CONFIGURATION OF THE HEART IN THE NORMAL ADULT

Angiocardiography has confirmed the usual concept of the formation of the cardiac contour except as regards the middle left segment. This segment, in the postero-anterior position, is defined as extending

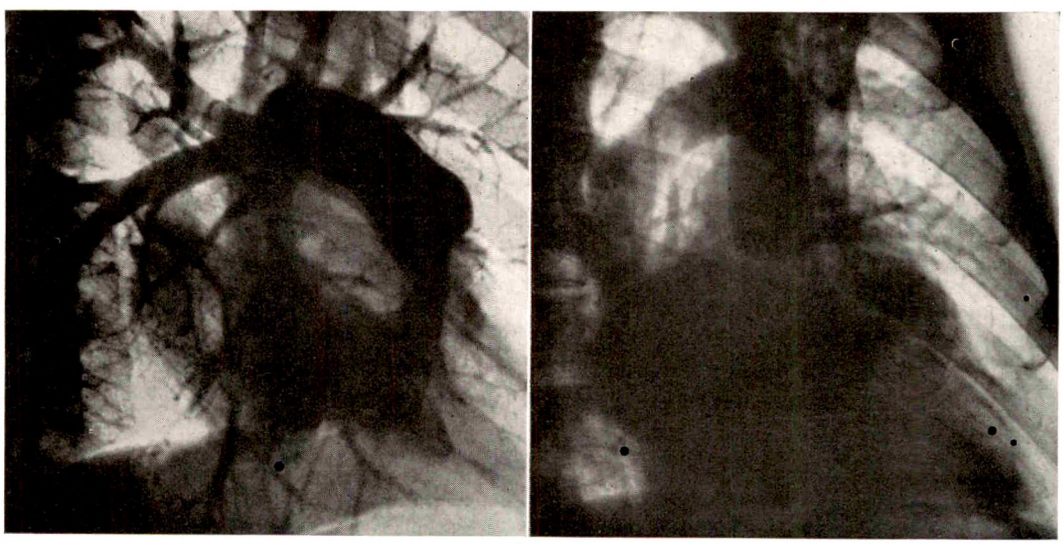


FIG. 2. Case 10. Angiocardiography made in the right anterior oblique position. Roentgenogram on the left shows right auricle, right ventricle and pulmonary artery.

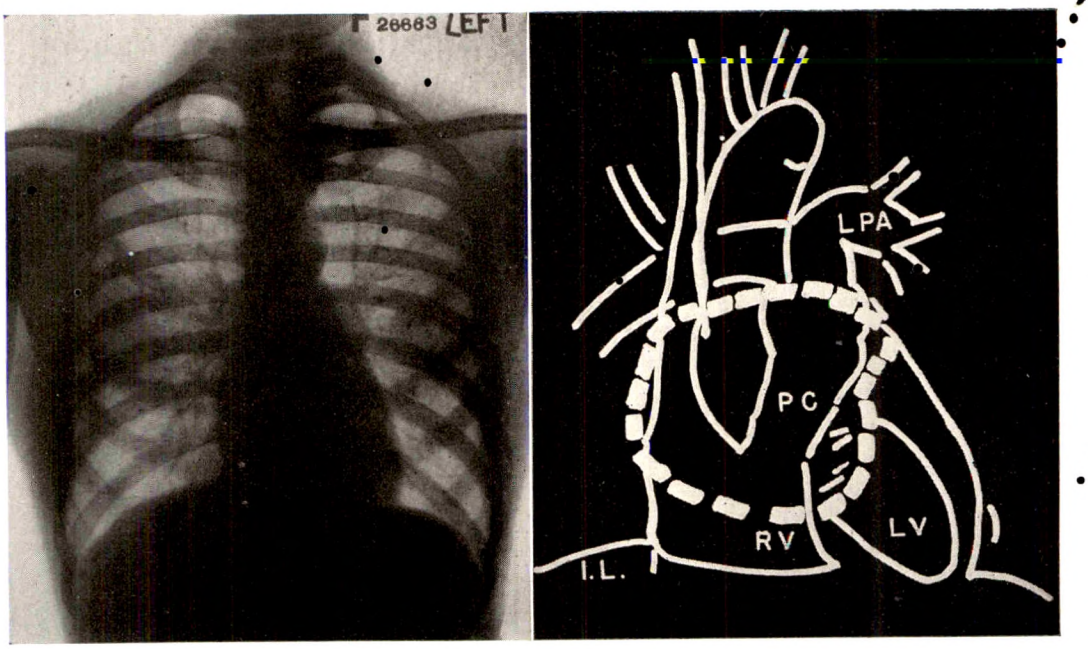


Fig. 3. Case 10. Mitral stenosis. Conventional posteroanterior roentgenogram and composite diagram of the findings illustrated in Figure 1.

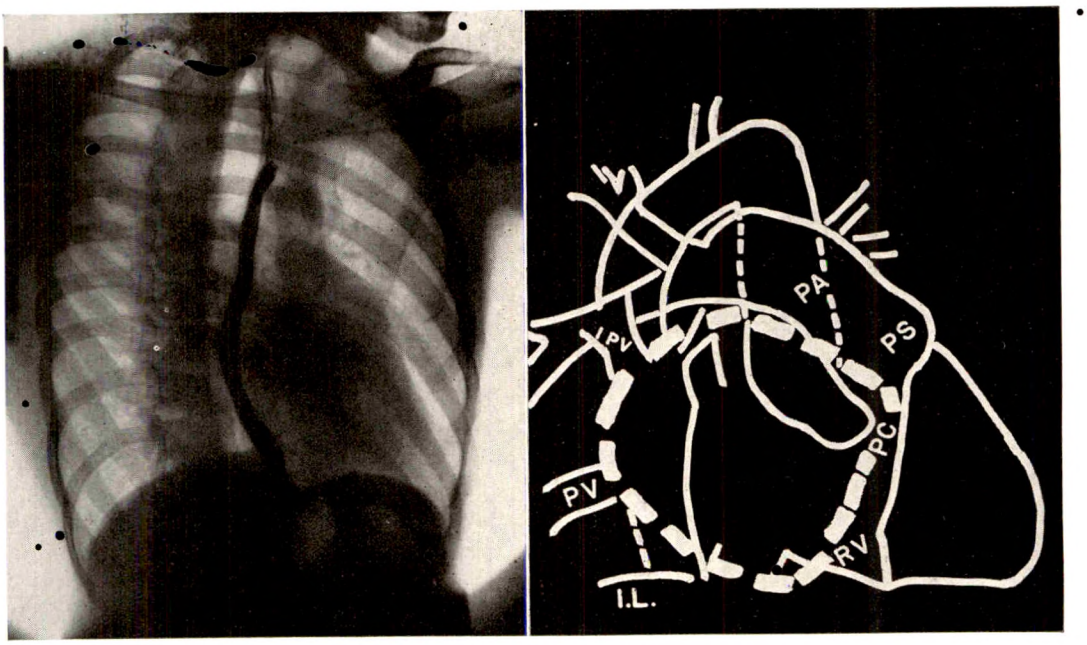


Fig. 4. Case 10. Mitral stenosis. Conventional right anterior oblique position with barium in the esophagus and composite diagram derived from Figure 2.

cephalad from a point about which "see-saw" motion takes place (point "G" of Vaquez and Bordet) to the junction of the contour with the aortic shadow. This has been described in the past as being formed by the left auricle (in some cases), pulmonic conus and pulmonary artery in this order passing cephalad. Angiocardiography has clearly shown that the pulmonary valve is situated well within the cardiac shadow. Hence, the pulmonic conus cannot possibly reach the cardiac contour. In fact, the middle segment has been found to be composed of the pulmonic artery with more or less of the left auricle caudad and sometimes by the edge of the main left pulmonary artery cephalad. Occasionally the pulmonary sinus produces a separate curve at the base of the pulmonary artery.

Our investigations confirmed the observation that the right cardiac border is made up of the right atrium in the normal individual. It is difficult to place the position of the right ventricle precisely, because only the cavity is opacified. However, the width of the ventricular muscle can be estimated and added. This usually falls well within the shadow of the opacified right atrium.

In the left oblique position, the usual analysis of the contours has been found correct. In this position also, the right border is composed of the right atrium. Where the border of the atrium joins the auricle a small notch may be seen. It is worthy of special note that the aortic valve and at least one inch of the ascending aorta are hidden in the cardiac shadow. The usual analysis of the right oblique configuration is also confirmed, except again that the pulmonic conus does not enter the left contour. The middle left segment in this position is composed of the pulmonary artery until the patient is turned into a marked oblique position.

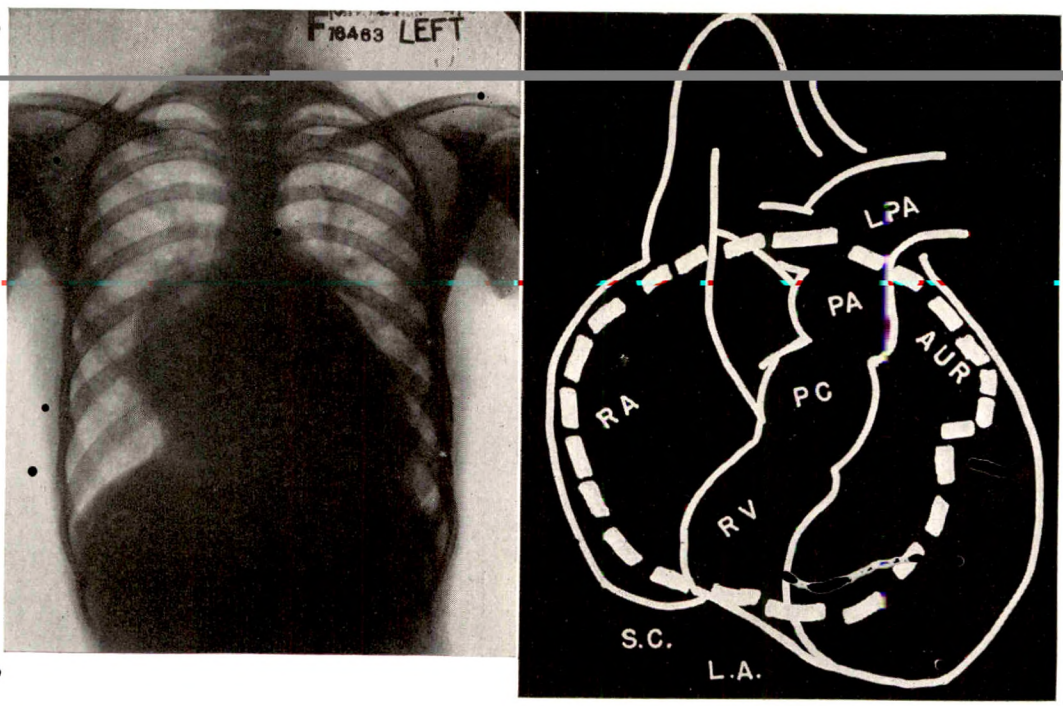


FIG. 5. Case 13. Mitral stenosis. Giant left auricle. Conventional posteroanterior roentgenogram on the left. Composite diagram on the right obtained from angiocardiography.

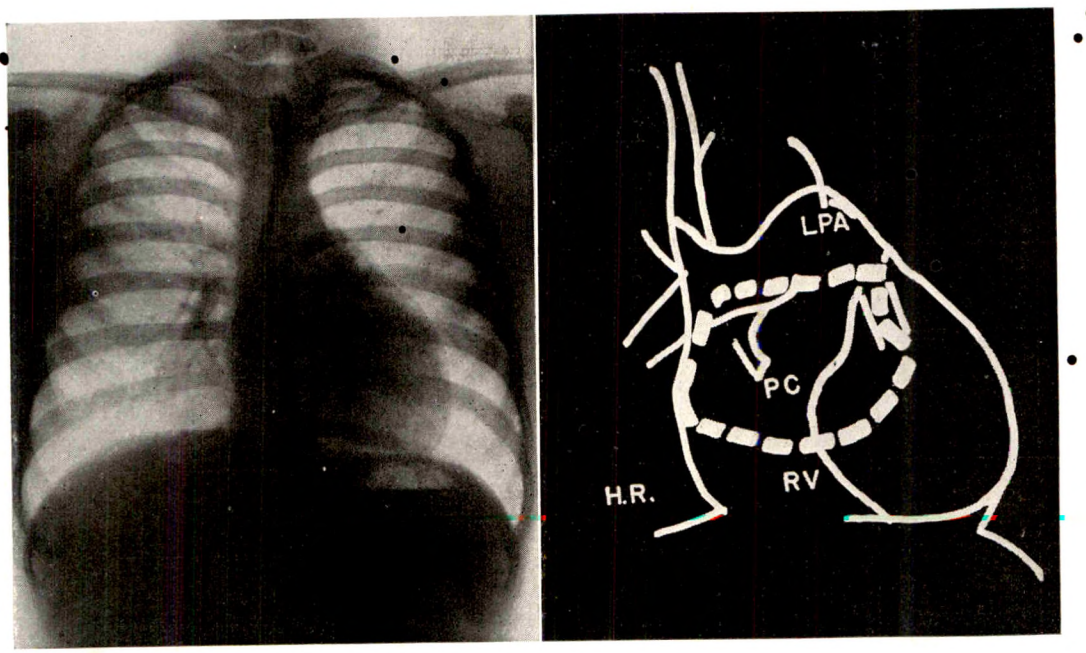


FIG. 6. Case 19. Mitral stenosis and insufficiency. Conventional posteroanterior roentgenogram with contour analysis obtained by angiocardiography.

MITRAL STENOSIS

Eleven cases with a typical mitral configuration were studied (Cases 1–11). These cases are particularly suited to angiocardiographic study because the altered dynamics in mitral stenosis results in stasis in

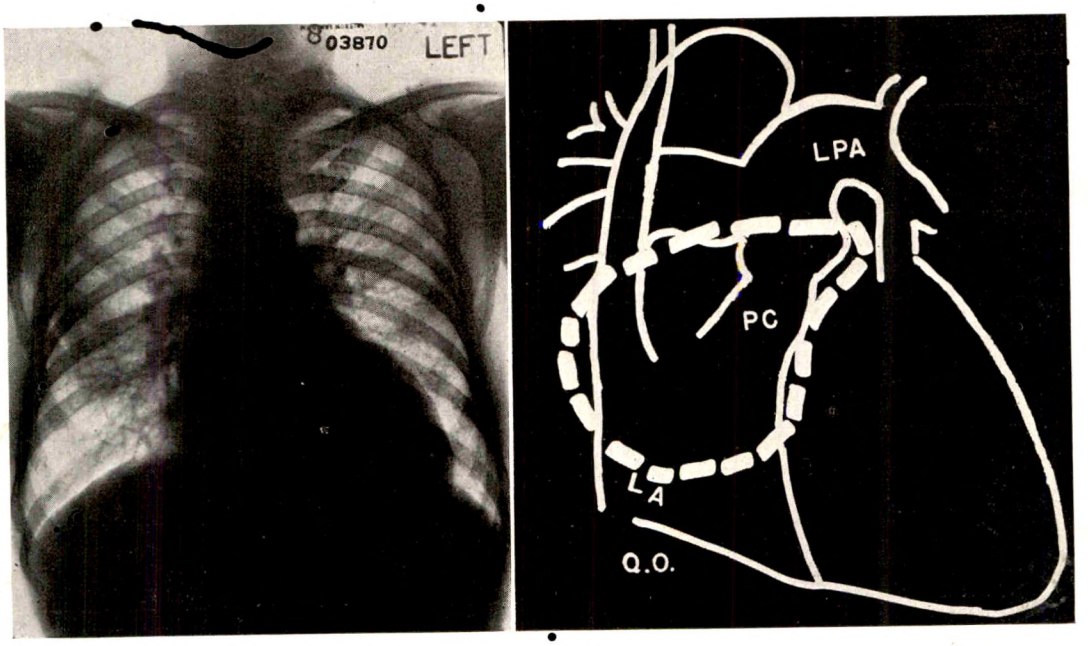


FIG. 7. Case 26. Predominant aortic insufficiency. Conventional posteroanterior roentgenogram with contour analysis obtained by angiocardiography.

TABLE I

Case No.	Clinical Diagnosis	Name	Sex	Age	First Attack Rheumatic Fever	Recurrence Rheumatic Fever	Heart Failure	Clinical Symptoms and Findings
1	Mitral stenosis; mitral insufficiency	A.C.	F	20	No rheumatic history. History of frequent colds	None	None	Moderate dyspnea on effort. Occasional cough with blood-streaked sputum. Murmur discovered at age of 10. Blood pressure 110/80
2	Mitral stenosis; mitral insufficiency	G.H.	F	39	Chorea at 13 yr.	Rheumatic fever at 17 yr.	At age of 36 yr.	Duodenal ulcer. No cardiac symptoms. Blood pressure 116/60
3	Mitral stenosis and insufficiency; tricuspid insufficiency	P.B.	F	35	12 yr.	None	24 and 35 yr.	Well up to 3 yr. ago followed by onset of auricular fibrillation. Marked right heart failure with pulsating liver. Blood pressure 112/78
4	Mitral stenosis and insufficiency	B.P.	F	40	14 yr.	32 yr.	At present	Palpitation on slight effort. Slight pretibial edema. Liver and lungs negative. Blood pressure 130/95
5	Mitral stenosis and insufficiency; aortic insufficiency	S.G.	M	16	8 yr.	15 yr. 16 yr.	At age of 16 yr.	Arthralgia and swelling of joints, epistaxis. Liver 2–3 cm. below costal margin. Systolic thrill over apex. Blood pressure 112/52
6	Mitral stenosis and insufficiency; aortic stenosis and insufficiency	D.I.	M	14	12 yr.	14 yr.		Sore throat, epistaxis, slight cough. Precordial pain. Blood pressure 90/50. Bilateral pleural effusion which disappeared following diodrast
7	Mitral stenosis and insufficiency	S.C.	F	39	10 yr.	None	32 up to present	Increased dyspnea on exertion. Progressive left and right heart failure. Marked hepatic enlargement. Venous distention. Systolic thrill over apex. Blood pressure 104/68
8	Mitral stenosis and insufficiency; aortic insufficiency	K.C.	F	30	3 yr.	11 yr.	None	Murmur discovered at age of 8 years. Asymptomatic at present
9	Mitral stenosis and insufficiency; aortic insufficiency	T.V.	M	19	Frequent colds as child	18 yr.	None	Murmur of mitral stenosis discovered at age of 15. Diastolic thrill at Erb's area. Pulmonary infiltration of undetermined etiology. Asymptomatic. Blood pressure 130/50
10	Mitral stenosis and insufficiency	I.L.	M	36	16 yr.	None	None	Symptoms due to duodenal ulcer. No cardiac symptoms. Presystolic thrill at apex
11	Mitral stenosis and insufficiency; aortic stenosis and insufficiency	J.C.	M	13	9 yr.	13 yr.	13 yr.	Otitis media at 12. This recurred with rheumatic fever at present admission. Development of dyspnea. Subacute bacterial endocarditis (*Streptococcus viridans*) briefly before discharge. Blood pressure 104/30
12	Mitral stenosis and insufficiency	A.M.	F	56	32 yr.	None	None	Sharp pain in left axilla aggravated by breathing for 2 mo. Moderate dyspnea on exertion. Blood pressure 120/60
13	Mitral stenosis and insufficiency	S.C.	M	16	11 yr.	15 yr.	15 and 16 yr.	Heart failure for past 1½ yr. Liver 6 cm. below costal margin. Systolic thrill at apex. Marked precordial bulge. Blood pressure 104/70
14	Mitral stenosis and insufficiency	S.P.	M	25	15 yr.	None	None	Patient had palpitation and dyspnea on exertion for the first time one year ago

Table I

Electrocardiogram	Phonocardiogram
Regular sinus rhythm, tendency to right axis deviation. P waves notched and prominent. RT2,3 slightly depressed, T3 diphasic. Tracing suggests right auricular and right ventricular enlargement. No digitalis	Systolic murmur over apex of low amplitude. Diastolic murmur over apex with presystolic accentuation. Opening click of mitral valve
Regular sinus rhythm, P waves wide and notched, RT2,3 slightly depressed, T3 diphasic. Tracing suggests auricular enlargement	Systolic and diastolic murmur over apex with occasional presystolic accentuation. Opening click of mitral valve
Auricular fibrillation. Tendency to right axis deviation. Small Q2,3. RT1,2,3 slightly depressed. T2 diphasic, T3 inverted. Tracings suggest right ventricular enlargement. Digitalis had been given	Early systolic murmur and diastolic murmur over the apex which starts after opening click of mitral valve. Murmurs are of very high frequency and moderate amplitude. On auscultation murmur had a musical character. Liver and venous pulses suggest presence of tricuspid insufficiency
Auricular fibrillation with slow ventricular rate. QRS slurred. Digitalis had been given	Faint systolic and prolonged diastolic murmurs best heard in the left parasternal area in the 5th intercostal space. Opening click of mitral valve over apex
Regular sinus rhythm. PR interval 0.24 seconds. P waves wide and notched. Tracing suggests auricular enlargement and impairment of conduction system	Faint systolic and prolonged diastolic murmurs best heard to the left parasternally. Opening click of mitral valve
Regular sinus rhythm. Tracing within normal variation	Systolic and prolonged diastolic murmurs over the apex. Systolic and early decrescendo diastolic murmur over Erb's and aortic areas
Regular sinus rhythm. Right axis deviation. P waves wide and notched. Small Q2,3. RT1,2,4 slightly depressed. T2,3,4 diphasic. Tracing suggests auricular and right ventricular enlargement	Systolic and prolonged diastolic murmur over apex with presystolic accentuation. Occasional opening click of mitral valve
Regular sinus rhythm. PR interval 0.22 second. P waves wide and notched. QRS high. Small Q2,3. T2 diphasic, T3 inverted. Enlargement of auricles and both ventricles.	Split first and second sounds at the base. Systolic and diastolic murmurs at apex with presystolic accentuation. Early diastolic of high amplitude and frequency at Erb's and aortic areas.
Marked sinus arrhythmia and bradycardia. P waves wide and notched. QRS high. Tracing suggests combined mitral and aortic disease. No digitalis	Early systolic and diastolic murmur at apex with presystolic accentuation. Opening click of mitral valve. Diastolic of decrescendo type at Erb's and aortic areas
Regular sinus rhythm. Peculiar axis deviation. P waves wide and notched suggesting auricular enlargement	Systolic murmur of low amplitude and diastolic of median amplitude over apex with presystolic accentuation. Occasional opening click mitral valve
Regular sinus rhythm. P waves wide and notched. QRS high and notched. RT1 slightly depressed. T1 diphasic. Tracing suggests auricular enlargement with enlargement of both ventricles. No digitalis	Prolonged systolic and diastolic murmurs over Erb's point, apex and aortic area. Opening click of mitral valve over apex
Auricular fibrillation. Right axis deviation. Small R4. RT2,3 depressed. T1 low. T3 diphasic. Tracing suggests enlargement of the right ventricle. Patient received digitalis	Prolonged systolic and diastolic murmur over 5th intercostal space to the left parasternally. Opening click of mitral valve
Auricular fibrillation. Frequent premature ventricular beat. Right axis deviation. QRS high =0.11 sec. RT1,2 depressed. T2,3 inverted. Tracing suggests enlarged right ventricle. Digitalis given	Systolic and prolonged diastolic murmur over apex. Opening click mitral valve
Auricular fibrillation. Tendency to right axis deviation. QRS =0.10 sec. RT1,2,3 slightly depressed. T1 diphasic. T2,3 inverted. Tracing suggests enlarged right ventricle. Digitalis given	Systolic murmur and opening click of mitral valve at the apex followed by short diastolic murmur

TABLE I—*Continued*

Case No.	Clinical Diagnosis	Name	Sex	Age	First Attack Rheumatic Fever	Recurrence Rheumatic Fever	Heart Failure	Clinical Symptoms and Findings
15	Mitral stenosis and insufficiency; aortic insufficiency; tricuspid insufficiency	T.W.	F	19	?	18 yr.	12 and 18 yr.	Innumerable admissions for heart failure for past 6 yr. Now admitted with marked orthopnea. Left recurrent paralysis. Marked hepatic enlargement
16	Mitral insufficiency	E.T.	M	17	Chorea at age of 16	None	None	Asymptomatic. Blood pressure 138/82
17	Mitral insufficiency	B.G.	M	13	?	None	None	Asymptomatic. Murmur discovered accidentally
18	Mitral stenosis and insufficiency	J.A.	M	15	Chorea 2 mo. ago	None	None	Chorea for 2 mo. No history of epistaxis or joint pains
19	Mitral insufficiency	H.R.	M	16	14 yr.	None	None	Asymptomatic
20	Mitral insufficiency	F.L.	M	17	? 14 yr.	None	None	Otitis media, upper respiratory infection and inflammation of symphysis pubis at age of 14. Now asymptomatic
21	Mitral insufficiency	S.H.	M	17	?	None	None	Frequent headaches. Occasional pain in right wrist. No cardiac symptoms
22	Aortic insufficiency; subacute bacterial endocarditis	B.Z.	M	44	8 yr.	None	None	Valvular lesion discovered at age of 18. Subacute bacterial endocarditis (*Streptococcus viridans*) for 3 mo.
23	Mitral stenosis and insufficiency; aortic insufficiency	J.B.	M	22	?	11 yr.	18 yr.; 22 yr.	Patient incapacitated for almost 3 yr. Marked systolic thrust in chest, neck and head. Liver 5 cm. below costal margin. No râles. Blood pressure 190/0?
24	Aortic stenosis and insufficiency; mitral stenosis and insufficiency	A.L.	M	30	?	None	None	Epistaxis, dizziness. Otherwise asymptomatic on rest and phenobarbital. Blood pressure 104/62. Symptoms suggested neurocirculatory asthenia
25	Aortic stenosis and insufficiency; mitral stenosis and insufficiency	F.L.	F	50	?	None	First attack 2 yr. ago	Onset of auricular fibrillation 1 yr. ago. Fever for 3 wk.
26	Aortic stenosis and insufficiency; mitral stenosis and insufficiency	Q.O.	M	55	?	None	50 yr.; 55 yr.	Murmur discovered 5 yr. ago. For 6 mo. increasing dyspnea with nocturnal paroxysms. Marked pulmonary edema. Enlarged liver
27	Mitral stenosis and insufficiency; tricuspid insufficiency; encapsulated pericarditis	F.M.	M	45	Arthritis at 31 yr.	?	37 yr.; 45 yr.	Engorged neck veins. Signs of right and left heart failure. Blood pressure 130/80. Venous pressure 8 cm. Saccharine time 33 sec.
28	Mitral stenosis and insufficiency; tricuspid insufficiency	M.B.	M	25	?	None	None	Pleurisy with effusion at 15 when murmur was discovered. Normal activity until 3 yr. ago. Progressive dyspnea for past 6 mo. Palpation, edema, pulsating liver, systolic thrill at apex

the left atrium with prolonged and intensified opacification. The middle segment of the left cardiac border could be divided, in the posteroanterior view, into an upper and lower arc except in one case. The cephalad arc was produced either by the main pul-

Table I—*Continued*

Electrocardiogram	Phonocardiogram
Regular sinus rhythm. PR interval 0.30 sec., suggesting impairment conduction system. RT2,3 slightly depressed	Systolic and prediastolic murmur at apex with opening click. Decrescendo early diastolic murmur over Erb's area. Pulse tracing suggests tricuspid insufficiency
Wenckebach's periods. First degree heart block after atropine. PR =0.30 sec. Otherwise within normal variation. Tracing suggests impairment of conduction system	Early systolic murmur "bruit de cannon" whenever PR is short
Regular sinus rhythm. Peculiar axis deviation. Otherwise within normal limits	Systolic murmur at apex. Maximum in mid systole
Regular sinus rhythm. Peculiar axis deviation. Otherwise within normal limits	Systolic murmur at apex of low amplitude. Opening click of mitral valve
Regular sinus rhythm. No abnormality	Systolic murmur at apex. Maximum in mid systole
Regular sinus rhythm. P waves notched. QRS low in lead I. Within normal limits	Systolic murmur at apex of low amplitude. Physiological third heart sound present
Regular sinus rhythm. P waves notched. Within normal limits	Early systolic murmur at apex. Physiological auricular and third heart sound present
Regular sinus rhythm. Left axis deviation. QRS 0.10 sec. RT2 slightly depressed	Systolic murmur at the apex. Early diastolic murmur of high frequency. Maximum over Erb's point
Regular sinus rhythm. Peculiar axis deviation. PR=0.26 sec. P waves very wide and notched. QRS =0.11 sec. T1 isoelectric. Tracing suggests auricular enlargement with myocardial damage. Patient received digitalis	Systolic and prolonged diastolic murmur of high amplitude over apex, Erb's and aortic areas. Right carotid pulse tracing showed anacrotic notch and vibrations in systolic portion
Regular sinus rhythm. Peculiar axis deviation. QRS high. RT2,3 slightly depressed. T2 diphasic. T3 semi-inverted. Tracing suggests combined right and left ventricular enlargement. No digitalis	First sound split. Systolic and diastolic maximum over the aortic areas
Auricular fibrillation. QRS high. RT1,3 slightly depressed. T1,3 diphasic. Tracing suggests combined right and left ventricular enlargement. Digitalis given	Systolic and prolonged diastolic murmur at apex. Systolic and early diastolic of decrescendo type at Erb's area
Regular sinus rhythm. Left axis deviation. P waves wide and notched. QRS slurred, wide and notched. RT1,2 slightly depressed. T1,2 diphasic. Tracing suggests auricular and left ventricular enlargement and myocardial damage. Patient received digitalis	Systolic and diastolic murmurs at the apex. Systolic of very high amplitude at the aortic area. Pulse tracing over carotid artery showed a low rise, systolic vibration and steepness indicative of aortic stenosis and insufficiency
Auricular fibrillation. Right axis deviation. Small Q2,3. QRS notched. T2 low. T3 inverted. Tracing suggests enlarge right ventricle with myocardial damage. Patient received digitalis	Heart sounds distant. Faint systolic murmur over apex. Pulsations of liver synchronous with arterial pulsation
Regular sinus rhythm. Marked right axis deviation. PR =0.25 sec. P waves wide and notched. Tracing suggests aurciular and right ventricular enlargement	Systolic and early diastolic murmur at the apex with opening click of mitral valve. Systolic and early diastolic murmur in tricuspid areas. Pulse tracing suggests tricuspid insufficiency

monary artery or by the left branch or both. The caudad arc was formed by the left auricle. In one case (Case 7) the entire middle segment was made up of the main pulmonary artery alone. The pulmonic conus was always found well within the car-

diac silhouette. In no instance did it contribute to the formation of the left cardiac border. The left atrium in every case formed the upper part of the right border.

The oblique views revealed that the enlarged left atrium displaces an elongated pulmonary artery anteriorly and cephalad producing the anterior bowing of these structures. The pulmonic conus and occasionally the entire right ventricle were displaced anteriorly. In this way also is explained the prominent anterior convexity of the mid left contour as it is visualized in the conventional right oblique position. In our cases, the pulmonary artery did not seem to be significantly dilated. Angiocardiography has established that the right ventricle does not enter into the cardiac contour. Certainly the prominence of the middle segment is not due to right ventricular enlargement primarily, but, as has been indicated, to left atrial enlargement with displacement and elongation of the pulmonary artery. The size of the right ventricle, therefore, cannot be estimated from the conventional roentgen examination. For purposes of comparison with the typical mitral configuration, the following groups also were studied:

Giant Left Atrium. Four cases of giant left atrium were examined (Cases 12–15). These presented no essential differences from the typical mitral configuration. In one instance, the giant left atrium extended far beyond the right atrium and formed the entire right border of the cardiac silhouette. In another instance, enlargement of the left atrium proceeded to the left rather than to the right and formed almost the entire left cardiac border.

Mitral Insufficiency. Six cases were studied in which the predominant lesion was mitral insufficiency (Cases 16–21). The cardiac configuration revealed a poorly defined small middle segment on the left border. The component parts, however, were the same as in typical mitral configuration. With a single exception, the left atrium showed insignificant enlargement.

Aortic Configuration. There were 5 cases of combined mitral and aortic disease which presented a typical aortic configuration (Cases 22–26). The middle segment of the left border was small and formed by the pulmonary artery or the left pulmonary artery or both. Below this, the left auricle occasionally approached the contour. In some instances, the enlarged left atrium participated in the formation of the right cardiac border.

Tricuspid Configuration. Two cases of triosteal disease (Cases 27–28) were studied. In both, the right ventricle was enormous but in neither did the pulmonic conus approach the cardiac border. The left middle segment was formed here, as in the other cases, by the pulmonary artery and mainly by the left auricle.

CONCLUSIONS

Angiocardiography has permitted an analysis of the formation of the cardiac contour during life. The method has been applied to the analysis of the mitral configuration. The enlarged left atrium, which is a constant finding in mitral disease, is the most important factor in the production of this configuration. It forms a considerable portion of the right cardiac contour. In addition, the left auricle forms the lower arc of the middle left cardiac contour. The upper or cephalad arc is formed by the pulmonary artery or the left pulmonary artery or both. The pulmonary artery is elongated and displaced anteriorly and cephalad by the left atrium and usually is not significantly dilated. The right ventricle does not enter into the cardiac contour. Its size is not proportional to the prominence of the middle left segment which is, in fact, produced indirectly by the left atrium, displacing the pulmonary artery. The pulmonic conus is always found well within the cardiac silhouette and does not contribute to the formation of the left cardiac border.

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NON-TUBERCULOUS PULMONARY CAVITATION

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EXPERIENCE has taught us that chronic pulmonary infiltrative lesions involving the upper lobes are predominantly tuberculous[2] and that when cavitation is present the diagnosis of tuberculosis is almost certain. In a hospital devoted almost exclusively to the care of tuberculosis, one would expect these criteria to be even more positive. Yet in an analysis of 2,000 autopsies at Sea View Hospital, on patients with cavitary lesions in the lungs, involving all lobes, Auerbach[1] has found approximately 1.5 per cent with non-tuberculous cavities. Since this percentage holds true at Sea View Hospital, it must be much greater in non-tuberculous or general hospital autopsy material.

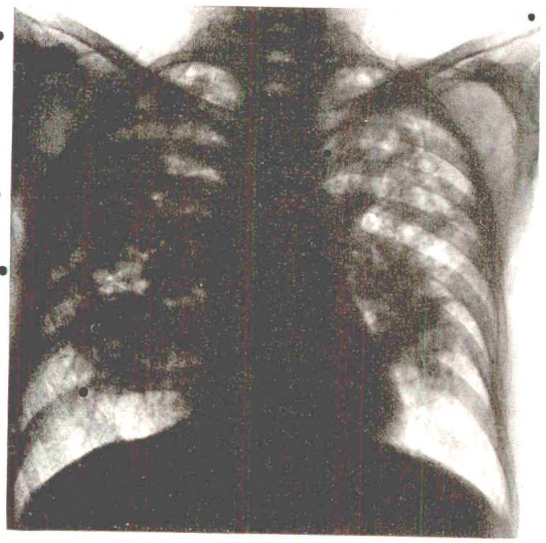


FIG. 1. Case 1. Anthracosilicosis. Extensive infiltration bilaterally, with a large irregular cavity just below the left clavicle. A large cavity is also visible in the right mid lung field. Autopsy diagnosis: anthracosilicosis with cavitation ("phthisis negra"). No evidence of tuberculosis was found.

It is the purpose of this paper to illustrate, roentgenographically, lesions of the lung which presented cavitation or what simulated cavitation, particularly of the upper lobes, and which on biopsy or autopsy proved to be non-tuberculous. The majority of these patients were referred to Sea View Hospital from other metropolitan hospitals, with the diagnosis of tuberculosis made clinically, roentgenographically, and in one instance apparently by a positive sputum examination.

The following cases illustrate some of the pulmonary lesions which may present cavitation of the lung fields roentgenographically, thus simulating tuberculosis:

REPORT OF CASES

CASE 1. C. S., white male, aged fifty-seven, with a twenty year history of hard coal mining and seven years' work with a jack hammer. Patient had a transient attack of asthma in 1925, about which very few details could be elicited. He was apparently well until 1936, when he entered Kings County Hospital because of cough, expectoration of black sputum, and dyspnea. Diagnosis on roentgen examination was chronic pulmonary tuberculosis; sputum was negative. He was discharged in December, 1936, but had a hemoptysis in July, 1937, and was readmitted to Kings County Hospital. He left the hospital after six weeks against advice.

Patient was admitted to Sea View Hospital on August 17, 1937, because of left chest pain and increasing dyspnea. Physical examination showed a chronically ill white male, markedly dyspneic, with slight cyanosis of the nail beds, barrel-shaped chest, diminished breath sounds throughout the chest with occasional moist râles in the left interscapular region.

Roentgenogram revealed a diffuse, bilateral, conglomerate infiltration with cavitation in the mid lung fields on both sides. There was evidence of a cavity with fluid level in the left upper lobe, just below the clavicle. Extensive emphysema was seen at both bases, with irregularity of the cusps of the diaphragm caused

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by adhesions. The trachea was deviated to the right side (Fig. 1).

The sputum and gastric examinations were persistently negative for tubercle bacilli and fungi. The sputum often contained black particles and was occasionally blood tinged. Patient expectorated about 2 ounces daily.

The hospital course was gradually retrogressive, with increasing dyspnea and marked wheezing. Patient died on December 6, 1937, apparently of respiratory failure.

Autopsy. The lungs showed anthracosilicosis of all lobes and multiple areas of cavitation involving the right upper, right lower, left upper and left lower lobes. There was no evidence of tuberculosis.

Comment. Schwartz and Auerbach[4] mention the fact that when, on the roentgenogram, cavitation is seen in a patient with a history of exposure to silica dust, there is a tendency to automatically make a diagnosis of silicotuberculosis rather than silicosis. This should never be done unless the sputum is positive for tubercle bacilli. "X-ray cavities in silicosis may be produced by emphysematous blebs, lung abscesses or necrosis of silicotic areas."

Case II. A. P., a female, aged forty-one, Puerto Rican, who was in comparatively good health until 1937, when she began to complain of cough, chills, fever, expectoration, and weight loss. She gave a history of having had influenza in 1921 and frequent upper respiratory infections since then.

Patient entered Bellevue Hospital the latter part of 1939, and a diagnosis of tuberculosis was made. She was placed on conservative therapy and was transferred to Sea View Hospital on September 3, 1940.

Admission roentgenogram at Sea View Hospital showed an enormous cyst in the upper half of the right lung, and numerous small cysts distributed throughout the remaining lower half of the lung. The right lung was shrunken and the right dome of the diaphragm was markedly elevated. The left lung showed no infiltration or consolidation and there was no evidence of any cavitation in the left lung (Fig. 2).

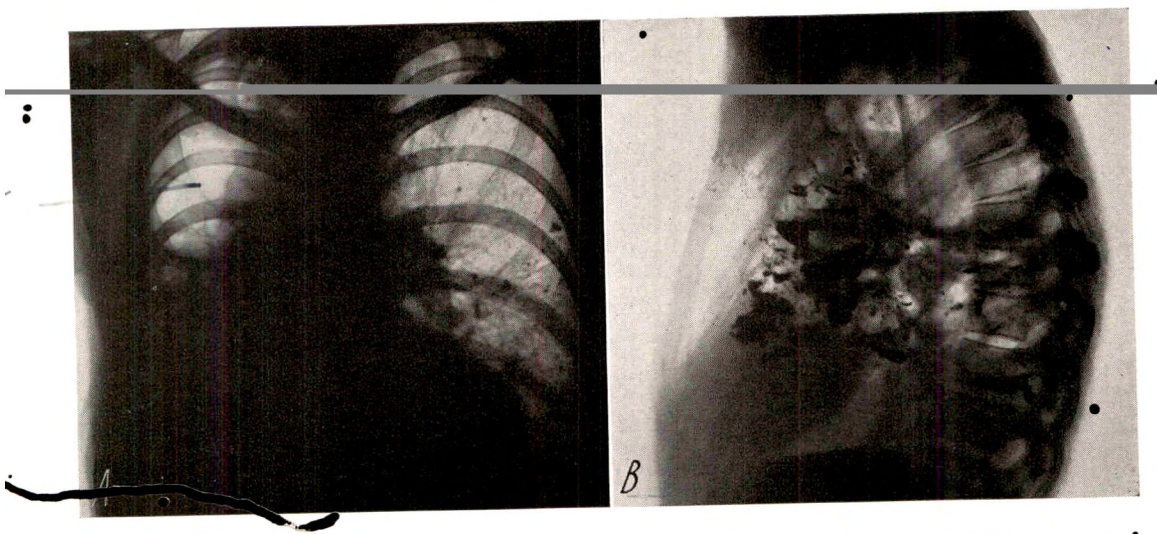


Fig. 2. Case II. Lung cyst. *A*, giant cyst in upper half of right lung field. Numerous smaller cavities are visible in right lower lung field. *B*, lateral view of right lung, following instillation of lipiodol. Huge cyst is visible in posterior portion of right upper lobe. Numerous bronchiectatic cavities are seen below the cyst. Lipiodol does not enter the cyst.

Examinations of the sputum for tubercle bacilli and fungi were persistently negative. Temperature ranged irregularly from 98° to 102° F.

Bronchographic examination showed multiple bronchiectatic cavities in the right lower and middle lobes. The lipiodol did not, however, enter the large cyst in the right upper lobe. The bronchi on the left side were normal. Bronchoscopy on December 4, 1940, showed a stenotic right main bronchus, and there was a soft, compressible mass, apparently rising from

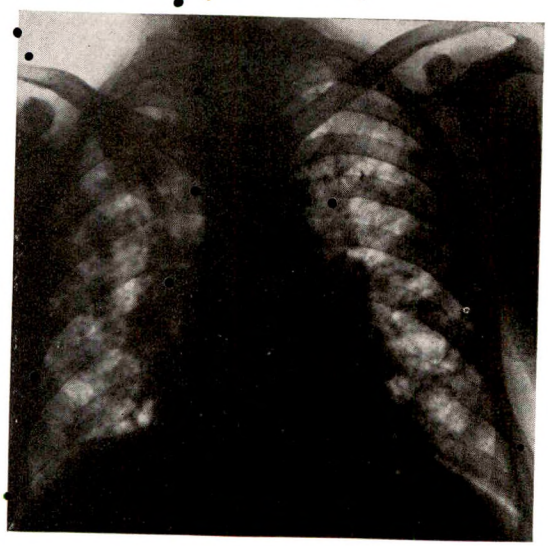


FIG. 3. Case III. Actinomycosis. Bilateral patchy type of infiltration with several highlights suggesting cavitation. Sputum positive for actinomyces, negative for tubercle bacilli.

the right wall of the carina, occupying the right middle lobe bronchus. Purulent secretions were seen coming from the stenotic opening. Removal of the mass was considered inadvisable.

A right pneumonectomy was performed on July 8, 1941. The opinion of the pathologist (Dr. Auerbach), after examination of the lung removed at operation, was that the large cyst in the right upper lobe was bronchiectatic in origin rather than congenital. The final diagnosis, therefore, was cystic bronchiectasis.

Comment. The thin wall, devoid of surrounding inflammatory reaction, in addition to the negative sputum, is important in the diagnosis of a lung cyst. Lung cysts may contain a small amount of basal exudate. The orifice of the communication with the bronchus is often so small that lipiodol cannot be introduced into the cavity.[5]

CASE III. L. D., white male, aged fifty-one, with a past history of exposure to silica dust as a coal miner for a period of about fifteen months. He had pneumonia on the right side in 1928 and on the left side in 1934. Pain developed in the left chest in July, 1938. He was admitted to Coney Island Hospital in October, 1938, where a diagnosis of bilateral pulmonary tuberculosis was made.

Patient was transferred to Sea View Hospital, where roentgen examination showed an exudative productive type of infiltration in the upper half of both lung fields, with several small highlights suggestive of cavitation (Fig. 3). Although the patient expectorated a box of sputum daily, no tubercle bacilli were ever found.

Bronchoscopy on October 26, 1938, was negative except for some reddening of the orifice of the right upper lobe bronchus. Smears of the bronchial secretions were negative for tubercle bacilli and fungi. Another bronchoscopy was done on March 1, 1939, at which time actinomyces were found in the secretions of the right upper lobe.

A chest wall abscess developed on March 30, 1939, and the aspirated pus was positive for actinomyces. The mass gradually disappeared. In June, 1939, the patient's condition became rapidly worse. His temperature began to spike daily, he became progressively more dyspneic, and died on June 25, 1939.

Autopsy. Examination at autopsy showed multiple actinomycotic abscesses of the lung, involving the left upper lobe, left lower lobe, and right lower lobe. There was also a small tuberculous excavation of the right upper lobe.

Comment. Detection of fungi in the sputum is the only decisive method of diagnosis. Sante[3] speaks of two forms of the disease: "One, peribronchial infiltrations clustering about the bronch of the hilum region and lower lobes; the other, forming large confluent granulating masses which ultimately break down, forming large abscess, or which extend to involve the pleural cavity, producing empyema."

CASE IV.* M. F., colored laborer, aged fifty-three, who was apparently well until May, 1939, when he noted increasing weakness, malaise, anorexia, weight loss and pain in the left chest radiating to the back. This was followed in a few weeks by a hemoptysis of 1,000 cc. of bright red blood. The patient entered a city hospital, in June, 1939, where a roentgen examination of the chest revealed the presence of an extensive area of increased density in the left mid lung field, and a large cavity with a fluid level, measuring about 5 cm. in diameter, present in the infiltrated zone (Fig. 4, *A* and *B*). One sputum

* This case was originally reported in full by Dr. Harry L. Katz, in the *Quarterly Bulletin of Sea View Hospital* 1940, *6*, 79–91.

examination was reported positive for tubercle bacilli. In view of this, the diagnosis of pulmonary tuberculosis was made and pneumothorax was instituted on the left in July, 1939. The sputum continued to be blood streaked for several weeks, but repeated sputum examinations were negative at that time for tubercle bacilli. Roentgenograms following the institution of pneumothorax revealed a visible mass in the left mid lung field, with moderate collapse of the lung. The Wassermann reaction was positive. Past history was non-contributory. He denied a luetic primary or secondary lesion, and he had never received antiluetic therapy.

On November 15, 1939, patient was transferred to Sea View Hospital for further therapy. Physical examination at that time showed a well developed but poorly nourished colored male. There was a lag in the left hemithorax, percussion note was impaired, and there were diminished breath sounds in the left chest in the upper half, anteriorly and posteriorly. Cardiac examination was essentially negative. Sputum was negative for tubercle bacilli and other laboratory findings were essentially negative.

A roentgenogram of the chest on November 17, 1939, revealed a rounded density, extending

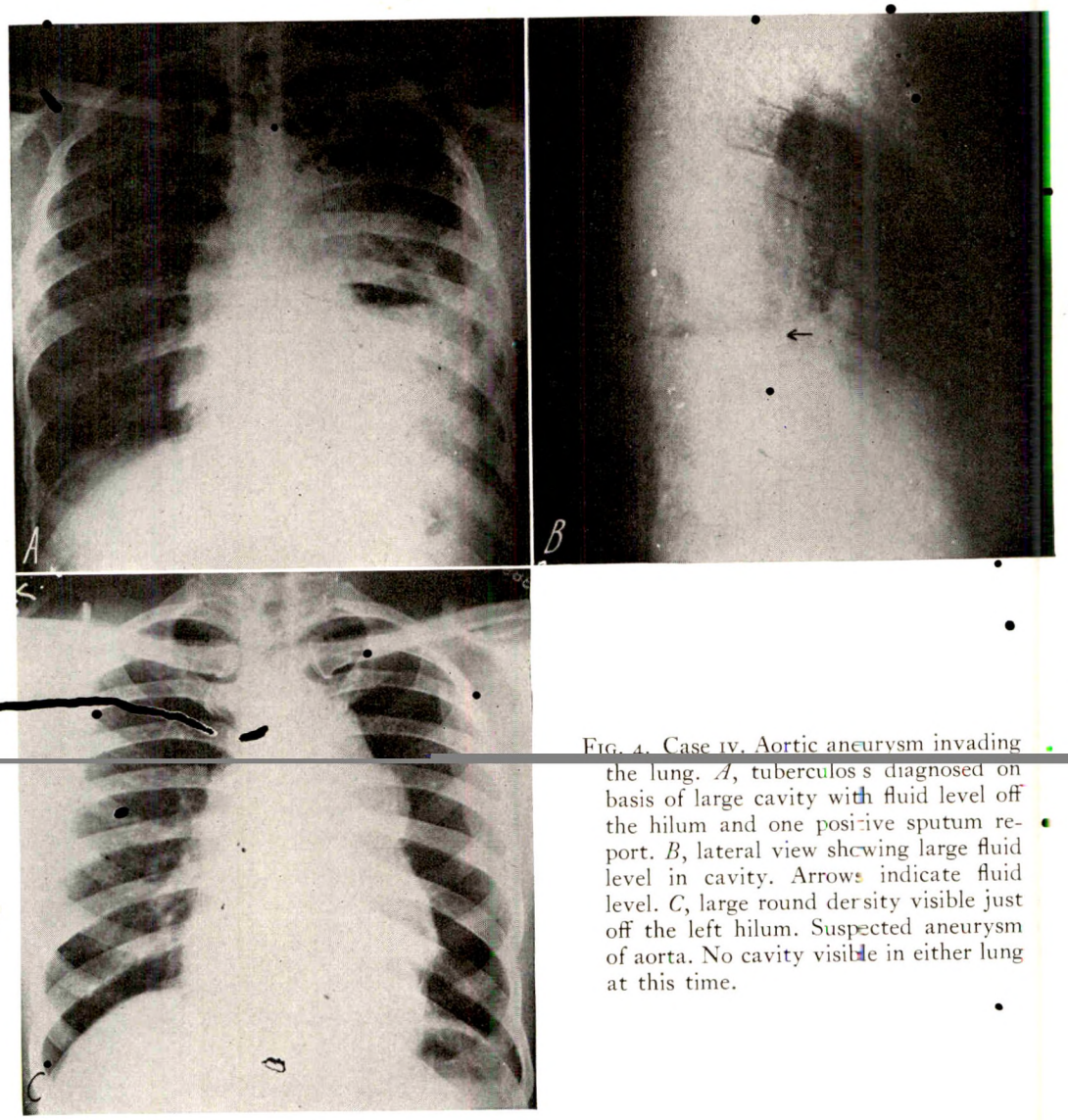


Fig. 4. Case IV. Aortic aneurysm invading the lung. *A*, tuberculosis diagnosed on basis of large cavity with fluid level off the hilum and one positive sputum report. *B*, lateral view showing large fluid level in cavity. Arrows indicate fluid level. *C*, large round density visible just off the left hilum. Suspected aneurysm of aorta. No cavity visible in either lung at this time.

laterally on the left side, fused with the shadow of the descending portion of the arch of the aorta, suggesting a localized aneurysm (Fig. 4, *C*). A roentgenogram of the spine showed no evidence of vertebral erosion. Bronchogram did not reveal narrowing or constriction of the left main bronchus by the aneurysm.

The patient received weekly pneumothorax refills of 100 to 150 cc. of air. On December 11, 1939, and again on December 13, 1939, he had hemoptyses of about 500 cc. of bright red blood. The sputum continued to be blood streaked for a considerable period of time and on January 24, 1940, he suffered a fatal hemoptysis of about 1,500 cc. of bright red blood.

Autopsy. It was found that there was a saccular aneurysm of the descending thoracic aorta which had invaded the left lower lobe and produced a large cavity, 5.5 cm. in diameter. There was no tuberculous involvement of either lung.

Comment. This case illustrates the danger of diagnosing pulmonary tuberculosis on a single positive sputum. A lung which shows a large cavity and yields persistently negative sputum after an initial positive should be suspected of disease other than tuberculosis.

CASE V. J. M., a white male, aged sixty, with a two months' history of weakness, weight loss, and cough productive of blood-streaked sputum. A roentgenogram of the chest showed a large cavity with basal fluid level in the left upper lobe (Fig. 5). The heart, trachea and mediastinum were shifted to the left, and the left leaf of the diaphragm was displaced upwards. Sputum examination was negative for tubercle bacilli.

Lipiodol studies showed obstruction of the left main bronchus. A biopsy obtained after bronchoscopy showed the presence of a bronchogenic adenocarcinoma of the lung.

Comment. In this case, the age of the patient, the brief duration of the symptoms, and the marked retraction of the heart and mediastinum with elevation of the diaphragm, serve to put us on guard against a diagnosis of tuberculosis. A chronic fibroid phthisis might give the same roentgenologic findings, but careful history would usually reveal a longer duration of symptoms than two months. Lipiodol frequently fails to enter cavernous neoplasms because the bronchus leading thereto is blocked by secretion, slough, pus or granu-

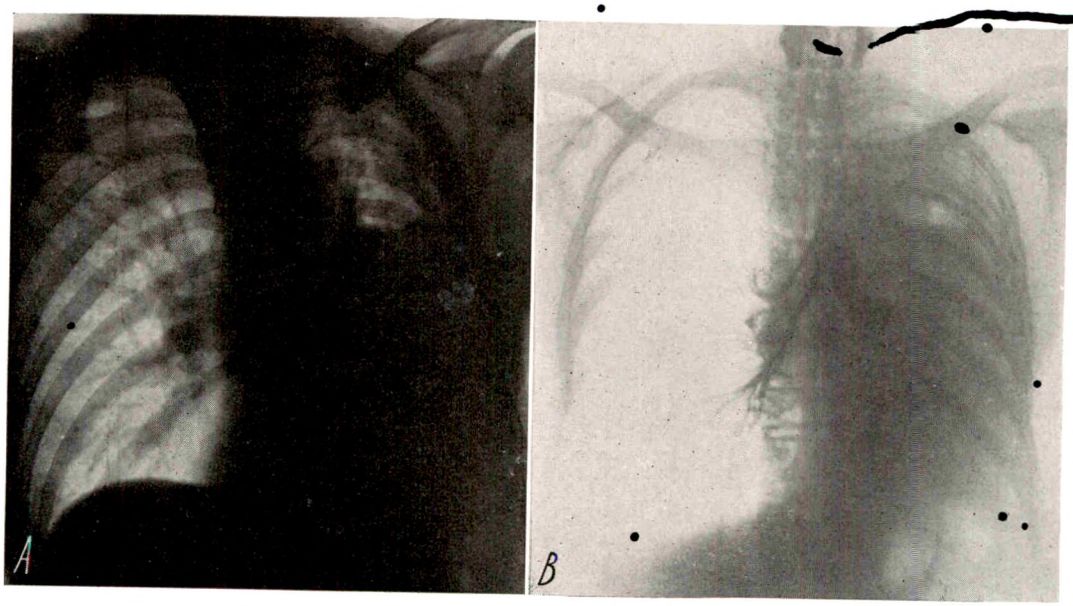


FIG. 5. Case v. Cavernous neoplasm. *A*, large cavity in left upper lobe, secondary to bronchogenic carcinoma. Heart and mediastinum are shifted to the left. *B*, bronchogram. Lipiodol is seen to stop abruptly at site of bronchial obstruction by carcinoma. Cavity with fluid level is seen in left upper lung field. Right bronchial tree is normal.

lation tissue, but the same is true of tuberculous or abscess cavities. The negative sputum and the biopsy were conclusive in making the diagnosis.

Case vi. A. T., Italian barber, aged fifty-six. His illness began in June, 1939, with a cough and expectoration which rapidly became profuse and foul. One month prior to onset, patient had several of his badly infected teeth extracted. A pain in the right upper chest was the first symptom noted. He was admitted to Greenpoint Hospital on June 17, 1940, with a chronic cough and profuse, foul sputum. A diagnosis of pulmonary abscess was made. Several bronchoscopies revealed pus from the right upper lobe. No tubercle bacilli were found in the sputum or gastric examinations.

Patient was transferred to Sea View Hospital on October 2, 1939. His general condition was fairly good. He expectorated 2½ cups of foul sputum daily, which was negative for tuberculosis but positive for streptococci, staphylococci, aerobes, anaerobes, fusiform bacilli, and spirochetes. A roentgenogram revealed a chronic lung abscess of the right upper lobe with a severe degree of surrounding bronchiectasis (Fig. 6). Bronchoscopy revealed thick, foul pus coming from the right upper lobe orifice. A pneumonotomy was done and the abscess drained, in one stage. A specimen of necrotic material was negative for neoplastic tissue.

Patient made an uneventful postoperative recovery. Sputum became scant and the odor disappeared. He gained 34 pounds in six months. The bronchocutaneous fistula slowly healed. A bronchogram on February 20, 1941, following the operation, showed residual bronchiectasis in the upper and mid lung fields on the right side. Patient's general condition was good and he was discharged on May 16, 1941.

Comment. Most lung abscesses are usually said to occur in the lower lobes. "The differentiation of a chronic lung abscess in an upper lobe from a tuberculous cavity may be very difficult, especially insofar as lung abscess, originally of pyogenic origin, may invade or reactivate an old tuberculous focus and even produce a positive sputum" (Sante). Repeated sputum examinations, however, will prove decisive in differentiation, since a tuberculous cavity of any size will persistently show many tubercle bacilli

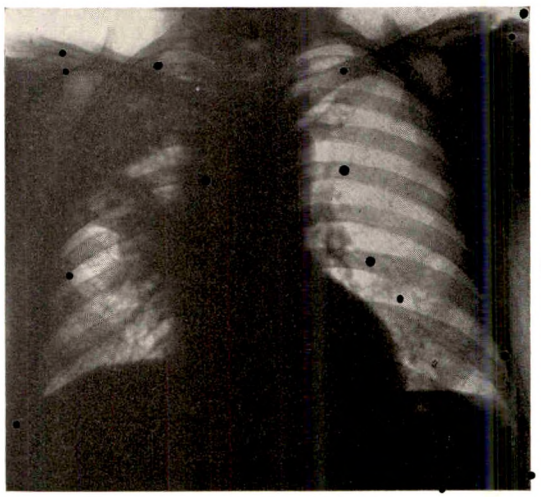


Fig. 6. Case vi. Lung abscess. Abscess cavity in right upper lobe. Sputum persistently negative for tubercle bacilli. Good result following surgical drainage of abscess.

on plain smear, whereas a pyogenic abscess invading an old focus will show only a rare bacillus in the concentrate.

Case vii. B. D., an Italian chauffeur, aged forty-five, transferred to Sea View Hospital from Kings County Hospital on July 28, 1941. Past history includes several bullet wounds from the World War in 1917, and operation for peritonsillar abscess in 1931. In March, 1941, he developed cough, productive of foul sputum, fever, weight loss and weakness. There was no hemoptysis. He entered Kings County Hospital, where sputum was persistently negative for tubercle bacilli. Diagnosis was "lung abscess, right upper lobe." He had a moderate hemoptysis in June, 1941, with streaking lasting for several days.

Patient was transferred to Sea View Hospital on July 28, 1941, where roentgen examination showed large abscess cavities in the right upper lobe (Fig. 7). The blood sedimentation rate was 125 mm. in one hour. Sputum was negative for tubercle bacilli, but contained fusiform bacilli, gram-negative bacilli and streptococci. There were no Friedländer bacilli or fungi. Examination of the sputum for cancer cells was also negative.

Bronchoscopy on January 14, 1942, showed copious foul purulent discharge from the right main bronchus. There was no evidence of obstruction in the bronchial tree. Because of the

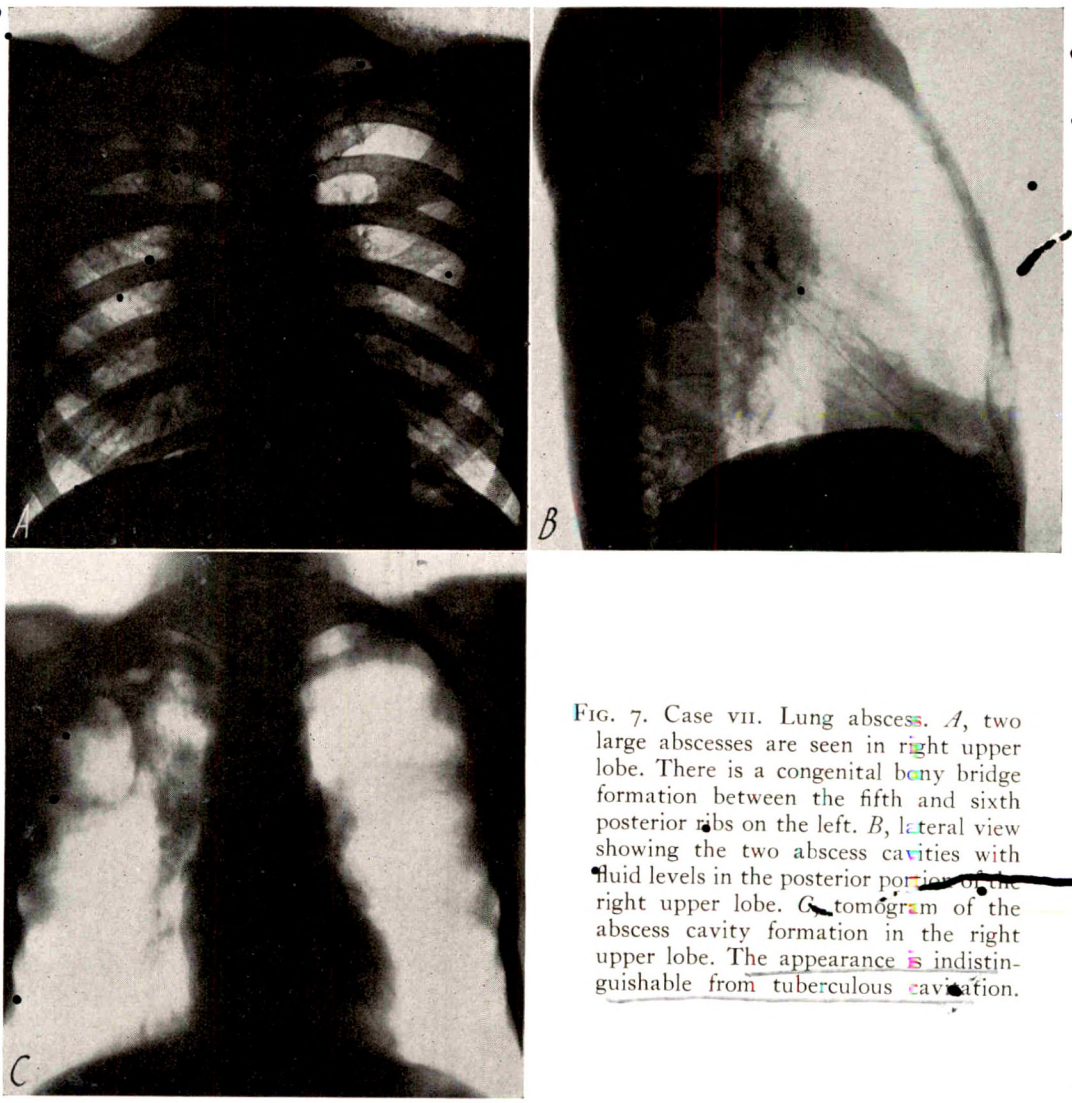


FIG. 7. Case VII. Lung abscess. *A*, two large abscesses are seen in right upper lobe. There is a congenital bony bridge formation between the fifth and sixth posterior ribs on the left. *B*, lateral view showing the two abscess cavities with fluid levels in the posterior portion of the right upper lobe. *C*, tomogram of the abscess cavity formation in the right upper lobe. The appearance is indistinguishable from tuberculous cavitation.

large size of the abscesses and their chronic character, it was the opinion of the surgical staff that pneumonotomy would not be of lasting value to this patient and that lobectomy, therefore, should be done. However, he improved somewhat on conservative therapy with postural drainage, and roentgen examination showed considerable decrease in the size of the abscess cavities. Patient ran a low grade temperature, with occasional rise to 101° F. In March, 1942, he left the hospital against advice.

Comment. In this case, there is no clear-cut etiology for the lung abscess and it is possible that we may be dealing with a chronic Friedländer bacillus infection. In a recent study of chronic Friedländer infections of the lungs, Solomon[6] found that the upper lobes were involved in 82 per cent of the cases and that lung abscess was the most frequent complication, occurring in 60 per cent of the cases. It is true that Friedländer bacilli were not found in the sputum in this case, but this is not conclusive. Solomon has pointed out that in the later stages of the disease, the organism is found mixed with or overgrown by secondary invaders.

Case viii. M.H., a white male, aged sixty-two, with history of cough and frequent blood streaking for almost twenty-seven years. At the age of fourteen, in Austria, he had afternoon fever, chills and frequent colds for a period of three to four months. He received no medical attention. Following this, he was well until about 1915, when he was admitted to Bellevue Hospital because of cough productive of blood-streaked sputum. There a diagnosis of bronchiectasis was made. Sputum was negative for tubercle bacilli at that time and has been negative to date.

In June, 1941, patient had a hemoptysis of about 400 cc. He was admitted to Sea View Hospital on June 27, 1941. Roentgen examination showed multiple areas of cavitation in the left upper lobe and considerable deviation of the trachea and heart towards the left, with tenting of the diaphragm and emphysema at the left base. Repeated sputum and gastric examinations were negative for tubercle bacilli. Bronchogram showed extensive saccular and cylindrical bronchiectasis in the left upper lobe (Fig. 8). Patient was afebrile and symptom free except for some morning cough.

Comment. Sante speaks of the "isolated cavity" type of bronchiectasis most frequently encountered in the hilar region. "The traction of scar tissue on the bronchial walls in diffuse fibrosis may be the etiological factor in the production of bronchiectatic cavities." Clinically, a case of bronchiectasis involving the upper lobes is indistinguishable from chronic pulmonary tuberculosis. The physical signs, the pro-

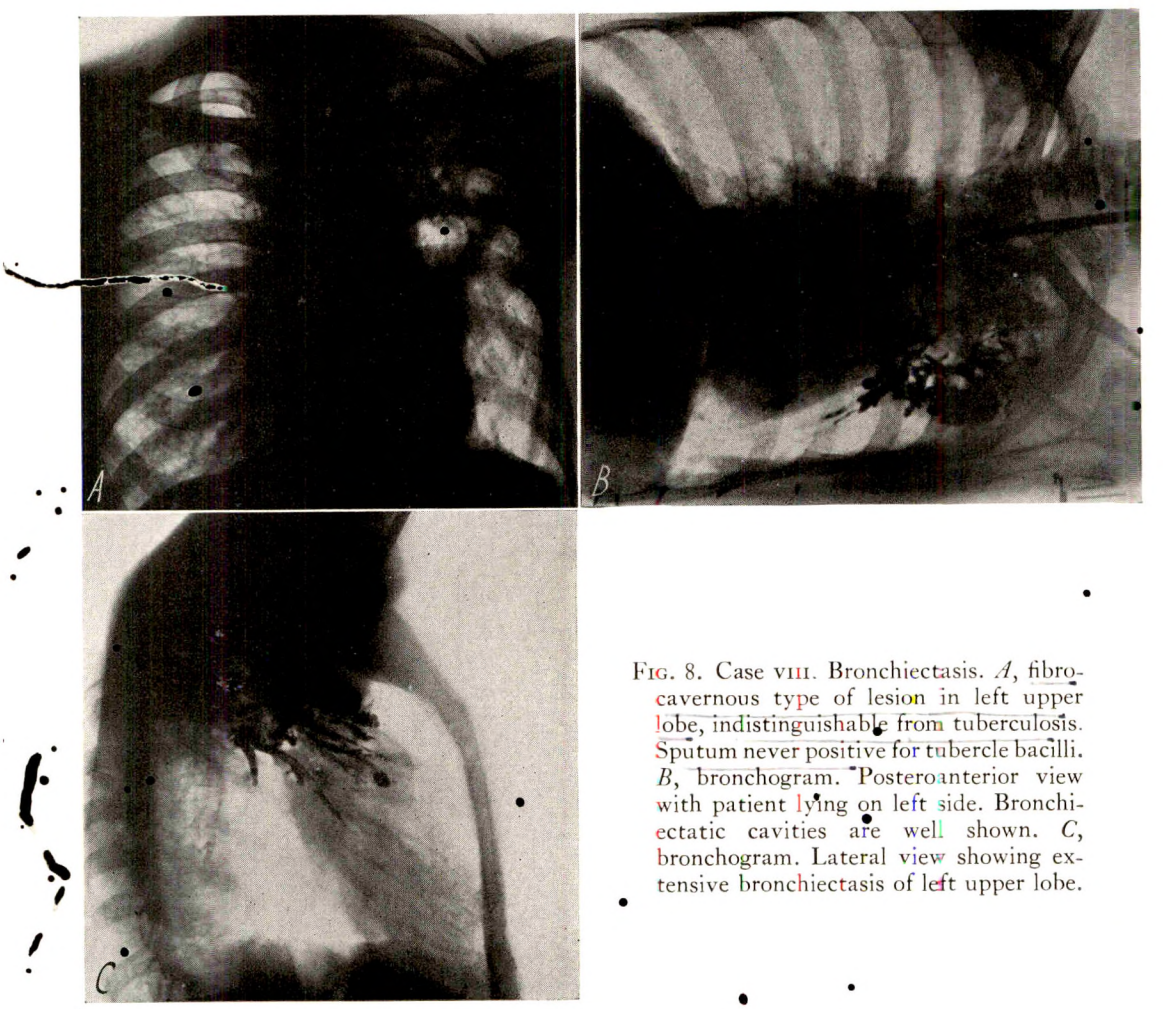


Fig. 8. Case viii. Bronchiectasis. *A*, fibrocavernous type of lesion in left upper lobe, indistinguishable from tuberculosis. Sputum never positive for tubercle bacilli. *B*, bronchogram. Posteroanterior view with patient lying on left side. Bronchiectatic cavities are well shown. *C*, bronchogram. Lateral view showing extensive bronchiectasis of left upper lobe.

false expectoration, the occasional hemoptysis are the same in both. "In distinguishing these cases from tuberculosis," according to Wessler and Jaches,[7] "the absolute limitation of the process to one lung is of the greatest importance. While fibroid tuberculosis may be restricted to one lung, usually, if the case is of some duration the roentgen examination will disclose some infiltration in the opposite lung."

CONCLUSIONS

Many pathological lung lesions will produce cavitation and simulate a cavitating tuberculous process. We have presented a series of cases from our material at Sea View Hospital illustrating some of these lesions. The majority of these patients were referred to our institution with a diagnosis of pulmonary tuberculosis, made clinically, roentgenographically and in one instance by a single positive sputum. The following chronic lesions are illustrated: anthracosilicosis, cystic disease of the lung, actinomycosis, aortic aneurysm producing pulmonary necrosis, bronchogenic neoplasm with parenchymal necrosis, a lung abscess of the upper lobe, cavitation probably as a result of Friedländer's bacillus infection of the lung, and a case of bronchiectatic cavitation of the upper lobe.

Other lesions will produce cavitation in the lung, and Winn[8] has recently reported twelve cases of pulmonary cavitation associated with coccidioidal infection. Slowly resolving nonspecific pneumonias may present areas of clearing that simulate cavitation and one may find cysts or bleb formation in association with pneumonias in children which will suggest a tuberculous lesion. Occasionally a gumma may cavitate centrally and simulate tuberculosis.

We must conclude, as others have done repeatedly before, that the roentgenographic diagnosis in itself is not conclusive, and that reports of series of this type are valuable in pointing out the possibility of other pulmonary lesions producing cavitation.

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Vol. 51, No. 1

EMPHYSEMATOUS CHOLECYSTITIS*

By C. A. STEVENSON, M.D.
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EMPHYSEMATOUS cholecystitis may be defined as an acute infection of the gallbladder characterized by gas production in the gallbladder lumen, walls, and pericholecystic tissues. Any virulent gas-producing organism may produce the disease when favorable local conditions exist.

Infection of the liver and biliary tract produced by gas-forming organisms has been recognized in many instances by surgeons and pathologists, but the roentgenologic demonstration of this condition is quite rare.

Hegner,[5] in 1931, reported what apparently was the first case in which it was possible to make a preoperative diagnosis of this condition by means of roentgenography. In fact, Hegner's case is the only one of its kind in the literature of this country, while Walters and Snell mention a similar case reported by Simon[11] in the French literature. Del Campo and Otero[2] of Uruguay have reported a case of emphysematous cholecystitis which was proved to be due to *B. welchii*.† Preoperative roentgenograms revealed gas in the biliary duct system, but a diagnosis of emphysematous cholecystitis was not made before surgical exploration. The infrequence of the roentgenographic diagnosis of emphysematous cholecystitis may be attributed primarily to the rarity of the disease itself and secondarily to the general lack of appreciation of the value of roentgenography in acute cholecystitis. Ramey and Scott[8] have shown that in a series of 110 cases of acute cholecystitis seen at the Scott and White Clinic from 1931 through 1940, roentgenographic examination of the gallbladder region, without any contrast medium, was of some value in the diagnosis of 40 per cent of the cases. In the cases reported by Hegner[5] and by Koch,[7] organisms closely resembling *B. welchii* were demonstrated in material obtained from the acutely inflamed gallbladders. Andrews and Henry[1], as well as other investigators, have shown that *B. welchii* is frequently present in the bile and walls of both normal and diseased gallbladders. Of the various microorganisms which have been found in the gallbladder and its walls, *B. welchii* is the one most likely to produce gas under anaerobic conditions. It is not likely that *E. coli* can produce gas in the gallbladder walls because of the lack of air or oxygen in this location.

Ramey and Scott, Walters and Snell, and other authors, believe that the first step in the development of acute cholecystitis is the lodging of a stone in the cystic duct. Their conclusions are based on the fact that from 75 to 96 per cent of gallbladders removed during the subsiding stages of acute cholecystitis will have stones in the lumen while almost 50 per cent will show a stone firmly wedged in the cystic duct. The most probable sequence of events in the production of emphysematous cholecystitis is: (*a*) lodging of a stone in the cystic duct; (*b*) resulting decrease in the local resistance of the gallbladder tissues allowing the avirulent *B. welchii* to become virulent; (*c*) increased dosage of the gas bacilli in the bile and gallbladder wall; (*d*) production of gas in the gallbladder, emphysematous blebs in the loose areolar connective tissue of the gallbladder wall, and extension of the infection to the pericholecystic tissues.

Emphysematous cholecystitis differs from other acute infections of the gallbladder in that the disease cannot be specifically identified by clinical examination but has a typical roentgenologic appearance. According to Walters and Snell, the disease terminates fatally as a rule.

† Now termed *Clostridium perfringens*, but will be referred to throughout this paper as *B. welchii*

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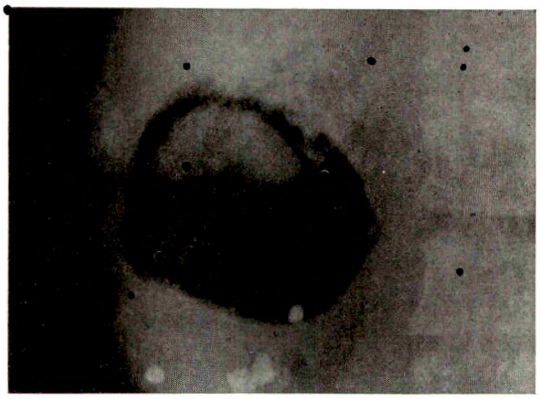


FIG. 1. Case 1. Gas in the gallbladder lumen. Uniform ring of gas in gallbladder wall and small irregular blebs just outside this ring of gas.

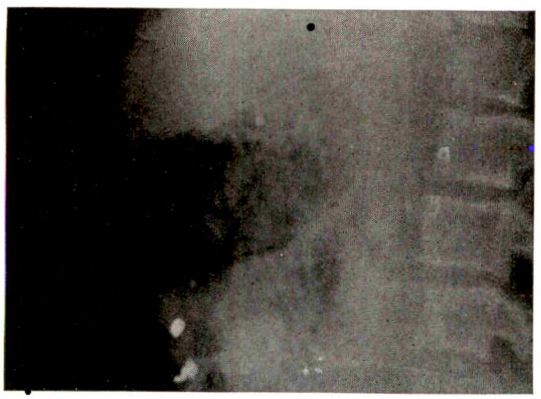


FIG. 2. Case 1. This lateral view shows the gaseous mass to be in the anterior half of the abdomen. The shape of a large gallbladder is easily recognized.

Three cases having roentgenologic findings similar to the case reported by Hegner are given here in brief.

REPORT OF CASES

CASE 1. J. A., male, aged sixty-four, was admitted to the University of Pennsylvania Hospital with a chief complaint of pain in the abdomen. The patient stated that he had known he had diabetes for the past fourteen years. In regard to his present illness, the patient said that eight days prior to entry into the hospital he noted some epigastric discomfort. Two days after that he began to have a dull pain in the right upper quadrant of his abdomen. The pain persisted, became more severe, and radiated to his right shoulder. On admission to the hospital it was evident that the patient, a well developed man, was acutely ill. Moderate jaundice of the sclera was discernible. A questionable mass was palpated in the right upper quadrant of the abdomen and there was moderate rigidity of the abdominal wall in this region.

Laboratory studies showed a leukocyte count of 13,000 with 86 per cent polymorphonuclear cells. Blood sugar analysis showed 190 mg. per 100 cc. The patient's temperature was 100.5°F.

A clinical diagnosis of acute cholecystitis, empyema of the gallbladder and diabetes mellitus was made. Roentgenologic investigation led to a diagnosis of emphysematous cholecystitis.

Figures 1, 2, 3, and 4 show the important findings.

The patient was operated on, and as soon as the omentum was opened a considerable quantity of cloudy fluid poured into the wound.

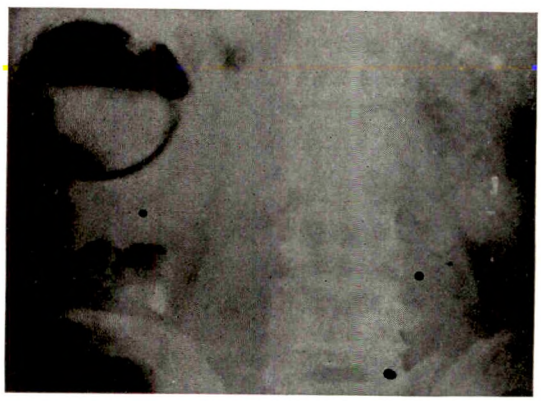


FIG. 3. Case 1. Patient erect. Note the intraluminal fluid level, gas above the fluid and gas in the gallbladder wall.

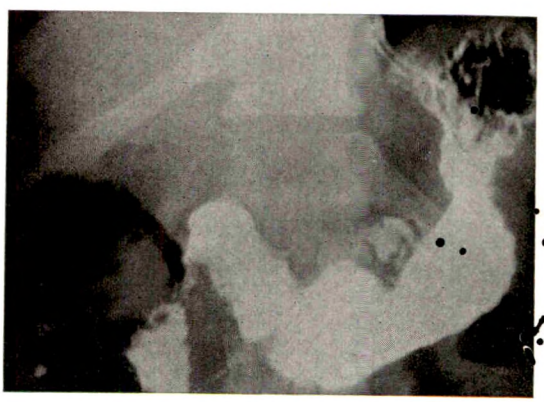


FIG. 4. Case 1. The duodenum is compressed by the large gallbladder.

After aspiration of this fluid, a completely gangrenous gallbladder, surrounded by a zone of gas, was found. A cholecystostomy was performed, and a large number of stones were evacuated from the gallbladder. Cultures were made of the peritoneal fluid only and showed aerobic and anaerobic streptococci, but no gas-producing organisms were found. The patient made an uneventful recovery and is now alive and well.

Case II. W. R., male, aged sixty-three, was admitted to a St. Louis hospital with a chief complaint of pain in the right side of the abdomen. Two days prior to his admission to the hospital the patient began to have a persistent dull pain in the epigastrium. The pain increased in severity and the next day it was localized in the right upper quadrant of the abdomen. Physical examination showed an elderly, obese man who was apparently acutely ill. No masses were palpable in the abdomen, but there was marked tenderness and rigidity in the right upper quadrant.

The patient's temperature was 102°F. and a leukocyte count showed a total of 26,250. A clinical diagnosis of acute cholecystitis was made but because of the patient's age, obesity, and poor general condition, he was treated symptomatically. The patient improved slightly over a five day period and on the sixth day a roentgenogram of the gallbladder region, without any opaque medium, showed a large gallbladder distended with gas, as is shown in Figure 5. Another roentgenogram made two days later showed that the gaseous outline of the gallbladder was lost and in its place was an irregular collection of emphysematous blebs surrounded by a gas filled, distended loop of small intestine (Fig. 6).

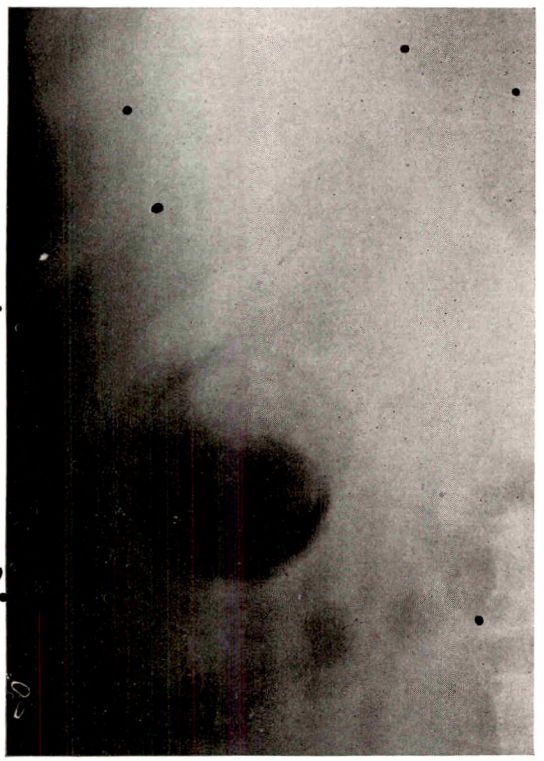


Fig. 5. Case II. Gas in the gallbladder lumen, walls, and pericholecystic tissues.

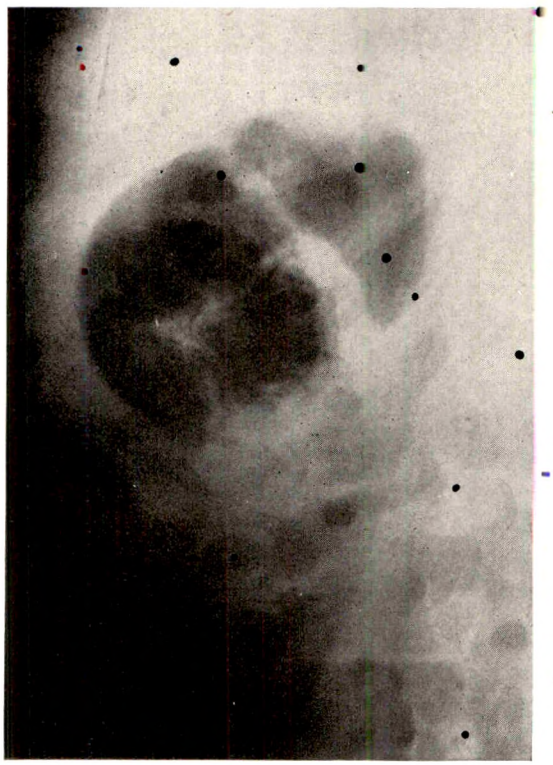


Fig. 6. Case II. The gaseous outline of the gall bladder wall is now lost. This is interpreted as meaning rupture of the gallbladder. A gas-filled loop of small bowel has surrounded the mass in an attempt to wall off the infection.

Fever and leukocytosis continued and gradually a large mass became palpable in the right upper quadrant of the patient's abdomen. On the eleventh day of hospitalization the patient was operated on. As soon as the peritoneum was opened there was a definite hiss of escaping gas. Large quantities of green pus welled up into the wound and about 400 cc. were removed by suction. A completely gangrenous gall-

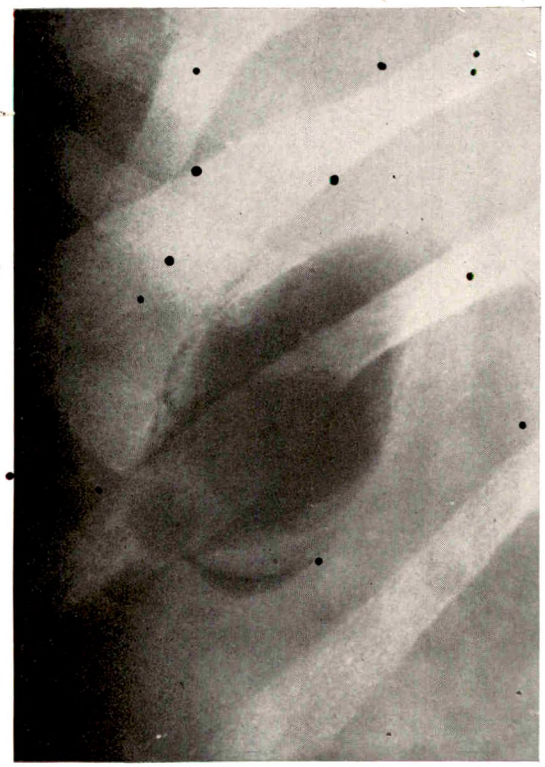


FIG. 7. Case III. Note the similarity of this gas-filled gallbladder to those of Cases I and II.

bladder was lifted out of the abdomen. There was a single stone, about 1.5 cm. in diameter, firmly lodged in the neck, and one wall of the gallbladder had perforated. Routine cultures of the peritoneal pus showed no bacterial growth. No studies were done for possible anaerobic organisms.

Except for a mild bronchopneumonia, the patient made an uneventful recovery and was discharged from the hospital on the twenty-second postoperative day. He is now alive and well.

CASE III. W. W., male, aged fifty-two, was admitted to the Scott and White Clinic with a chief complaint of pain in the abdomen. For the preceding five years the patient had had occasional attacks of anorexia, intolerance to fried and greasy foods, and discomfort in the right upper part of the abdomen. Five days prior to admission to the clinic, the patient experienced a sudden severe pain in the epigastrium and right upper part of the abdomen. The pain persisted and soon nausea and vomiting developed. In spite of symptomatic treatment the patient steadily became worse.

Physical examination showed a well developed, slightly obese man who was apparently acutely ill. A moderate icteric tint of the skin

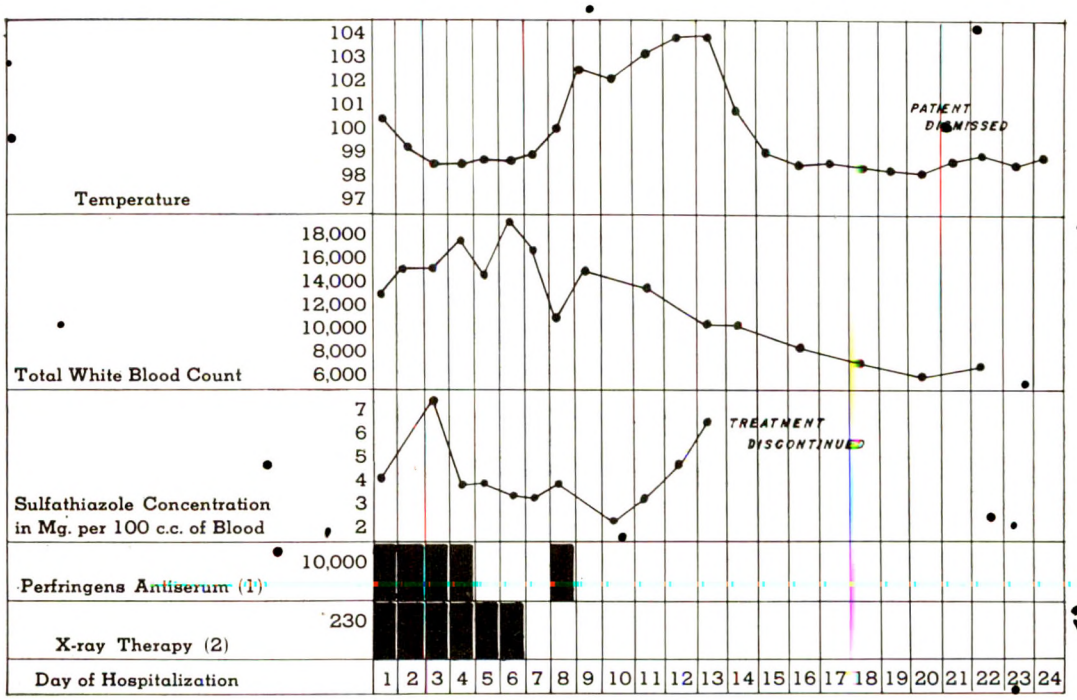


FIG. 8. Case III. This graph shows the relationship between the medical treatment and the patient's temperature and total white blood count.

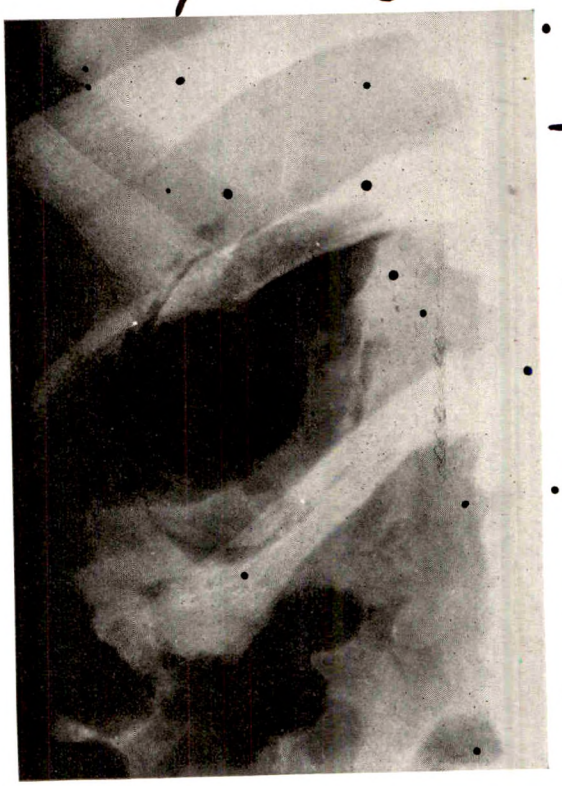


Fig. 10. Case III. Third day. Note large, emphysematous bleb superimposed on the gallbladder shadow.

was noticed and a tender mass about 10 cm. in diameter was palpable in the gallbladder region. The patient's temperature was 100.5°F., and the total leukocyte count was 13,000.

A clinical diagnosis of acute cholecystitis was made, and a roentgenogram made on the

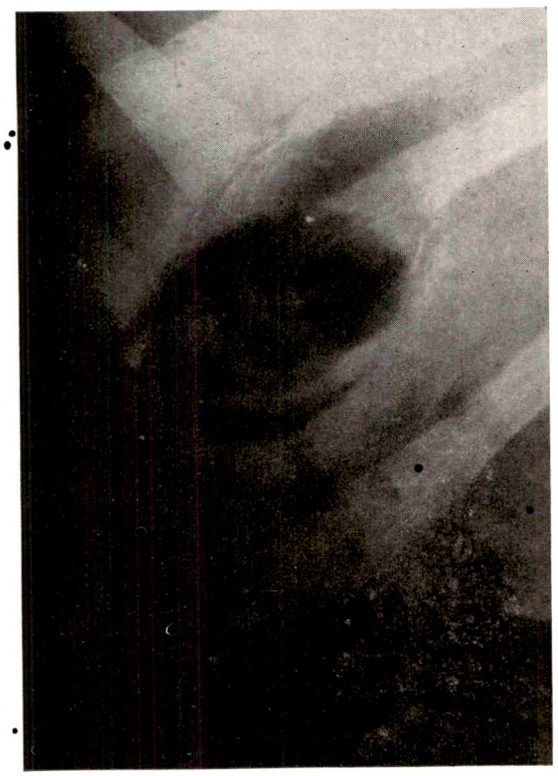


Fig. 9. Case III. Second day of hospitalization. Note increase of gas in gallbladder wall.

day of admission to the clinic led to a diagnosis of emphysematous cholecystitis (Fig. 7).

Recognizing the work of Kelly and Dowell[6] in the use of roentgen therapy for gas gangrene, the well-known value of specific antiserum in gas gangrene, and the possibility of favorable action of sulfathiazole,[4] a combination of all these therapeutic agents was used in the treatment of this patient.

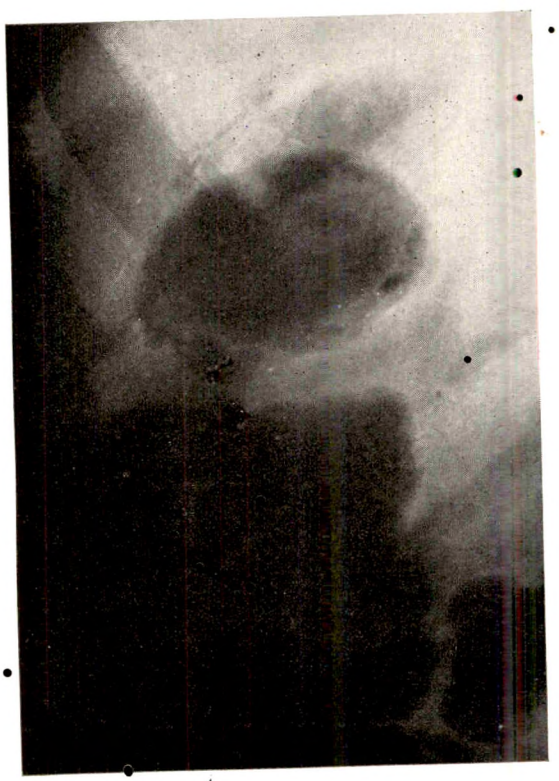


Fig. 11. Case III. Fourth day. Slight decrease in amount of gas in gallbladder wall.



FIG. 12. Case III. Definite decrease in amount of gas in the wall on the fifth day.

Figure 8 is a composite graph showing the relationship of the treatment to the patient's temperature and total leukocyte count.

Serial roentgenograms showed a definite correlation between the amount and extent of the gas in and around the gallbladder to the patient's general condition (Fig. 9–16).

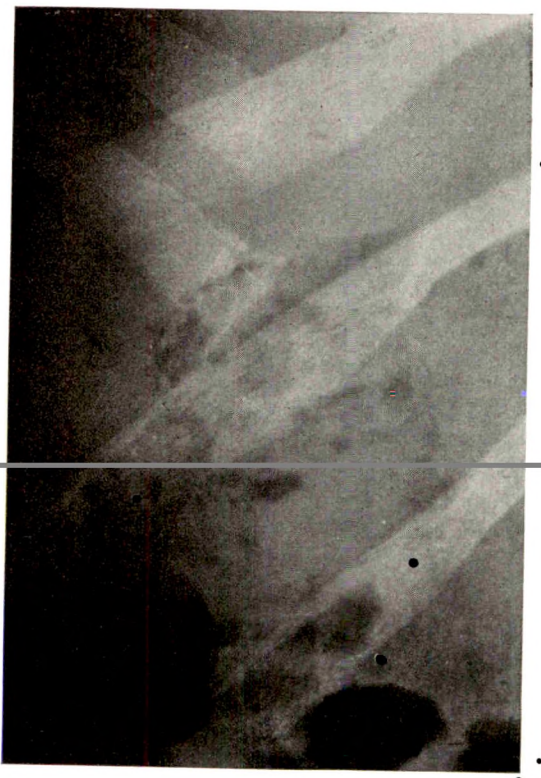


FIG. 14. Case III. Eleventh day. Decrease of gas in all areas.

After six days of this medical treatment the patient improved markedly. A sudden rise in temperature after this improvement was thought to be due to either a late serum reaction or excess sulfathiazole concentration in the blood. The temperature promptly returned to normal when the sulfathiazole was discontinued. The patient was dismissed from the hospital on the twenty-fourth day.



FIG. 13. Case III. Eighth day. Marked decrease in the gas in the gallbladder lumen.

The patient has been seen recently and is now in excellent health. Roentgenographic examination of the gallbladder, after administration of the radiopaque dye, showed no evidence of concentration of the dye, no radiopaque stones and no abnormal gas shadows. Roentgenologic investigation of the stomach, duodenum, and colon showed no abnormal findings.

SUMMARY

Cases I and II were proved to be emphysematous cholecystitis but unfortunately no intensive effort to demonstrate *B. welchii* was made. Case III had identical roentgenologic findings but since the medical treatment was successful, no proof was obtained.

All three cases showed gas in the gallbladder, blebs in the wall, and collections of gas in the pericholecystic tissues. No gas was noted within the biliary duct system. These findings are identical to the roentgenograms of the case reported by Hegner.

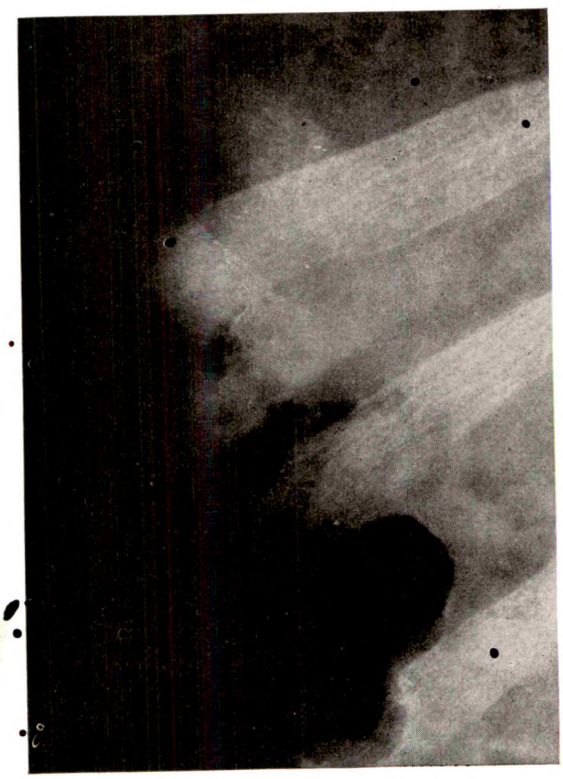


FIG. 15. Case III. Sixteenth day. Only a few small blebs remain. Patient clinically well at this time.

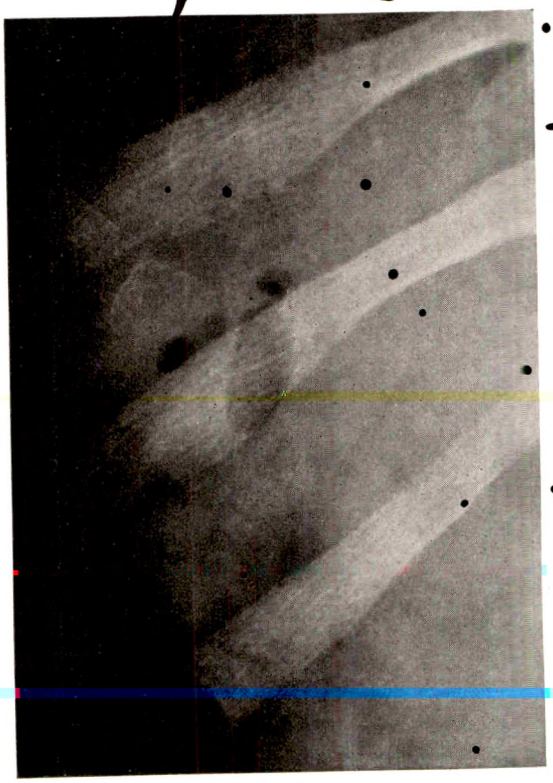


FIG. 16. Case III. Twentieth day. Three blebs remain. Patient was dismissed from the hospital four days after this roentgenogram was made.

There was no evidence of gas gangrene elsewhere in any of the four patients. The presence of gas in the gallbladder lumen alone is not sufficient for the diagnosis of emphysematous cholecystitis for cases of biliary fistulae commonly show gas in the gallbladder either with or without gas in the biliary duct system.[12]

It is interesting to note that Hegner's patient and the three patients described here were men, whose average age was sixty-three years in those operated on, while the patient receiving medical treatment alone was fifty-two years old. In the cases surgically proved, no patient gave a history suggesting previous gallbladder disease while the patient receiving medical treatment evidently had intermittent attacks of cholecystitis for about five years.

The previous idea that emphysematous cholecystitis is invariably fatal is disputed

by the fact that the three patients described here are now alive and well.

I am greatly indebted to Dr. E. P. Pendergrass, Professor of Radiology at the University of Pennsylvania, for his kindness in furnishing the history and roentgenograms of Case I as presented in this paper.

Dr. H. R. Senturia of St. Louis, Missouri, is due credit and appreciation for furnishing the history and roentgenograms of Case II.

CONCLUSIONS

1. Three cases of emphysematous cholecystitis are added to the literature.
2. All three cases had similar roentgenologic findings and two of the cases were correctly diagnosed preoperatively.
3. Medical treatment consisting of sulfathiazole, *B. welchii* antiserum and roentgen therapy was successful in the treatment of one case.
4. Roentgenograms of the gallbladder region are of distinct value in cases of acute cholecystitis. The roentgenographic demonstration of gas in the gallbladder lumen, emphysematous blebs in the gallbladder wall, and collections of gas in the pericholecystic tissues is indicative of acute gangrenous cholecystitis, most likely caused by *B. welchii*.

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VOL. 51, NO. 1

ROENTGEN THERAPY FOR BRONCHIOGENIC CANCER*

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ROENTGEN therapy in bronchiogenic cancer is a universally accepted procedure of treatment. The result is palliation and these benefits are widely acknowledged. There is considerable variance of opinion as to whether life is actually prolonged by irradiation.

This investigation, therefore, has been an attempt to estimate the longevity cycle as well as correlate the factors of dosage which might be shown to have some relationship to the more favorable results in a series of 167 cases of bronchiogenic cancer receiving irradiation. The longevity cycle of irradiated cases was compared with another series of 119 cases which received no treatment. These were all microscopically proved cases. Autopsy confirmation was obtained in 53 per cent of the entire group. The roentgenologic interpretation was approximately 65 per cent correct. Bronchoscopic examination was by far the most important diagnostic procedure.

A review of the literature indicates that the incidence of primary bronchiogenic cancer has risen at an accelerating rate, being about 10 per cent of all cancers. This frequency is comparable to cancer of the large intestine. The present knowledge of the nature and consequences, as well as statistical analyses of the incidence, pathologic classifications and concomitant symptoms, is discussed in detail in the splendid monograph by Simons,[18] the symposium by Halpert,[7] and many other contributions, notably by Ochsner and DeBakey,[13] Jaffé,[9] Dublin,[4] Menne and Anderson,[12] and Matz.[11] Our own findings are essentially in accord with these other studies.

Lymph node biopsy was done in 7 cases. Pleural effusion revealed cancer cells in 19 instances. Lobectomy was done in 2 cases and surgical explorations in 3 cases. Roentgenologic investigations for skeletal metastases were not done routinely, except in cases where there was a clinical suspicion of such involvement. There were 11 cases with metastasis to the spine. Rib erosion was relatively common as seen in routine chest examinations in late cases.

The distribution of bronchiogenic cancer (100 cases) was as follows: in the right lung, main bronchus, 22 per cent; upper lobe, 27 per cent, and lower lobe 9 per cent; in the left lung, main bronchus, 19 per cent; upper lobe 15 per cent, and lower lobe 8 per cent. Until the development of surgical techniques, as well as experiences conclusively demonstrating a possible curability by radical surgical removal, the treatment of primary cancer of the lung has been regarded as a radiologic procedure. Unfortunately, the majority of the cases are advanced and inoperable when a clinical, roentgenologic or bronchoscopic diagnosis is made. There are, likewise, many patients who are already too ill for any kind of treatment when the diagnosis is made. It is obvious why it is so important to continue on with irradiation procedures in the vast majority of cases, because the only alternative would be to do nothing.

The roentgenologic criteria of operability are not always accurate, except for the circumscribed or peripherally located tumors. These types also offer diagnostic difficulties, particularly for bronchoscopic procedures. Our own experience with aspiration of tissue for microscopic study has been discouraging. The possibilities of metastatic cancer, cysts, neurofibroma, fibrosarcoma, hilar and mediastinal lymph node tuberculosis, aneurysm, empyema and lymphoblastoma, as well as other pathologic entities must be considered.

Explorations for diagnosis or surgical re-

* Presented at the Forty-Third Annual Meeting, American Roentgen Ray Society, Chicago, Ill., Sept. 14–18, 1942.

moval may be an ideal procedure but permission for such intervention is not always obtainable nor does the physical condition always permit such an approach.

Graham, Singer and Ballon[5] conclude that irradiation does not prolong the lives of patients with cancer of the lung because the great majority of the tumors are radioresistant.

Overholt and Rumel[16] studied the life expectancy of inoperable cases and found, on the average, that the patients who received high voltage roentgen therapy lived only two-thirds as long as untreated patients. Overholt[15] believes radiotherapy shortens the life of patients with cancer of the lung.

Portmann[17] believes that irradiation may afford symptomatic relief but does not prolong life. Craver, on the basis of 178 cases, reports that radiation therapy not only showed symptomatic relief but also prolonged life.

Alexander[1] feels that the only proper use of high voltage roentgen therapy is as a therapeutic test for possible lymphoblastoma and for palliation in inoperable, malignant neoplasms. He believes[2] that the use of roentgen therapy for other purposes is useless and may even be dangerous, because of the loss of valuable time, except for palliation in inoperable cases.

Steiner[19] did not find that survival was notably prolonged by irradiation; 21 irradiated patients survived for an average of 11.9 months after the onset of symptoms, as compared to 10.5 months in 53 control patients.

Leddy and Moersch[10] reported 25 of 125 patients alive one year or more after treatment; 3 were alive 119, 127 and 146 months respectively after starting treatment. Of 125 untreated patients none survived longer than one year. Since 25 patients who were treated lived from one to twelve years, the absolute value of roentgen therapy as a palliative measure for bronchiogenic cancer was regarded as self evident. Leddy and Moersch regard some of these results as so-called cures.

Tenzel[20] found that the survival period of irradiated patients was on an average five months longer than that of patients not so treated. The average duration of life from the onset of symptoms to death for the irradiated patients was fifteen months, and for those not given irradiation only ten months. Twice as many irradiated as non-irradiated patients survived more than one year.

Goldman[6] reported data on 11 patients with epidermoid carcinoma in which the duration of life was two years or more. Two of these were irradiated and were still alive 84 and 244 months after onset of symptoms but lived only 24 and 4 months respectively after the diagnosis was established. Nine patients lived 28, 30, 53, 61, 75, 98, 120, 151 and 168 months without any kind of treatment. Of these 9 patients, 2 did not live a month after the diagnosis was made; 2 lived one month; 1 lived three months; 1 lived four months and three lived 7, 11 and 12 months respectively.

It is a problem to decide when the malignant process actually started. The manifold variety of symptoms of cough, pain, dyspnea, etc., the unusually long duration of years in many instances by comparison with the very short duration of days or weeks in other cases creates a situation of considerable confusion and uncertainty when evaluating the life cycle of a bronchiogenic cancer. Results are poor because of the advanced stage of the disease at which a correct diagnosis is made in the majority of cases.

It is important and interesting to point out that many cases were clinically and roentgenologically regarded as primary cancer of the lungs, and found at autopsy to be Hodgkin's disease, lymphosarcoma, neurofibroma, tuberculosis and particularly metastatic cancer (Table 1). The sulcus tumors of Pancoast are considered here to be of bronchiogenic character.

There is no uniformity of opinion as to the histopathologic classifications of bronchiogenic cancer. The confusion seems to arise in proportion to the degree of ana-

plasia. The microscopic report was "carcinoma" in 37 per cent of the cases; squamous cell carcinoma in 28 per cent; adenocarcinoma in 24 per cent of cases; 11 per cent of the group were the so-called basal or reserve cell, round cell, spindle or oat-shaped cell types.

Steiner studied the microscopic effects of roentgen therapy on primary cancer of the lung, and in 21 cases irradiation of the tumor with doses up to 5,000 r did not destroy the cancer as judged by microscopic standards.

Difficulties of diagnosis exist even with the aid of bronchoscopy and comparative roentgenograms. Unresolved pneumonia, persistent suppuration or any indeterminate roentgenologic appearance should be considered as a possibly malignant neoplasm until proved otherwise. A peripherally located tumor may be slow growing and when symptoms develop the growth may be shown roentgenologically to involve an entire lobe. Cancer complicated by infection may result in many confusing roentgenologic appearances, and these changes vary widely according to the duration of the disease. Holman and Pierson[8] reported convincing evidence of cancer of the lung simulating roentgenologic effects of inflammatory disease. At certain stages in the development of pulmonary cancer there may be complicating infection with resulting characteristics of an intrapulmonary abscess, of purulent bronchitis, of bronchiectasis or even of tuberculosis.

Menne and Anderson[12] describe two macroscopic types. One, in which the major lesions, at least in the less advanced stages, seemed to be that of maximum hilar accumulation, the hilar nodular type; another, in which there was a diffuse involvement with a minimum of hilar concentration was termed the diffuse, necrotic type.

Olds and Kirklin[14] believe that abnormal, unilateral, hilar infiltrations are sufficiently constant to be regarded as almost pathognomonic, roentgenologic evidence of bronchiogenic cancer. This interpretation is in accordance with the results of this study.

TABLE I

AUTOPSY FINDINGS IN CASES ROENTGENOLOGICALLY CONSIDERED AS BRONCHIOGENIC CANCER (NOT INCLUDED IN THIS SERIES)

	No. of Cases
1. Metastatic Cancer Arising in:	
(a) Brain	1
(b) Esophagus	2
(c) Kidney	1
(d) Rectum	1
(e) Prostate	1
(f) Breast	3
2. Mediastinal Tumor:	
(a) Lymphosarcoma	–
(b) Hodgkin's disease	3
(c) Neurogenic tumor	1

Atelectasis was also noted frequently, and varied according to the stage of the disease when investigations were instituted. Pleural effusion occurred in 57 cases. Lung abscess, bronchiectasis and tuberculosis were frequent complications revealed at autopsy.

The technical procedures of irradiation varied considerably. A voltage of 200 kv. was the same throughout. The filtration was almost always 0.5 mm. of copper, but in some cases 2 mm. of copper was used. The target skin distance was uniformly 50 cm. Treatments were usually given every second day. Two, three and four fields of entry were irradiated on the same day. In a few cases cross fire technique was used with four fields anteriorly and four fields posteriorly. The majority of the cases were treated with one field directed through the anterior chest, and one field through the posterior chest. Various sizes of fields were used, as follows: 6 by 8 cm., 10 by 10 cm., 15 by 20 cm., and 20 by 20 cm.

The dose never exceeded 200 r given to each of two, three, four and occasionally more fields on the same day at the rate of three treatments each week. In some instances daily treatment was given according to the "Coutard method." The total doses are shown in Chart 1 as tissue doses (r) at the approximate tumor level, and the total on the skin varied according to the size of the field and the thickness of the individual.

The tissue dose at the approximate tumor level was 2,500 r in the majority of cases. The predetermined plan of dosage was usually 3,500 r, but treatments frequently were discontinued because of failing health and strength. All doses that were less than 2,500 r were treatments discontinued because of progressive weakness.

More than half of the treated cases received in excess of 2,500 r. This experience

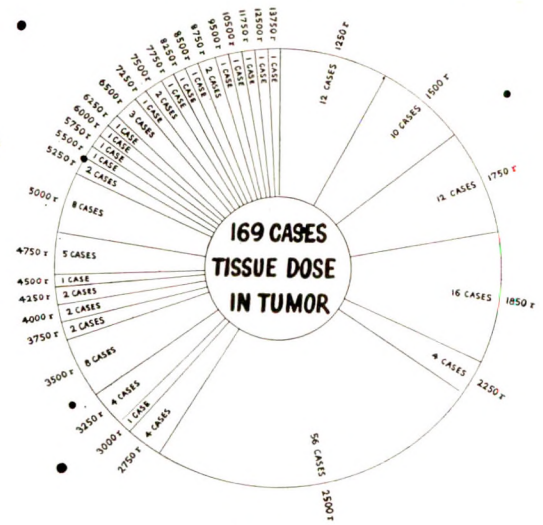


Chart I

revealed that the skin and physical tolerance may permit total doses of 8,000 r, 10,000 r, and 12,000 r. Any treatment that exceeded 2,500 r represented the total dosage of two, three or four series given at six, eight and twelve week intervals. In a small number of cases serial roentgenograms showed undoubted evidence of complicating, irradiation fibrosis, even after a single series of 2,000 r. These changes have been discussed by me elsewhere,[21] and are regarded as an effect of the underlying cancer and sometimes complicating infection.

Intrabronchial insertion of radon in gold seeds was done in 2 cases. Thoracotomy with direct insertion of radium into the peripherally located tumors has not been done in this series of cases. Radium packs were used in 9 cases. The dosage ranged from 5,000 to 30,000 millicurie-hours.

The problem of determining the longevity cycle is not simple. Histories were often vague as to the onset of the first symptom. In the majority of instances this evidence seemed clear, but the latitude ranged from a few days to several years or more. The average duration of symptoms in 114 cases where the case histories seemed to be very definite was 8.9 months. There were 2 cases with a history of cough extending over a period of twenty years. Eleven cases had some symptoms longer than three years.

Cancer of the lung was frequently an accidental finding when investigations were instituted to determine vague symptoms of fatigue, loss of weight, general indisposition and sometimes gastrointestinal complaints. Frequently the pulmonary infiltration was extensive when the study was made within a few days or few weeks after the first symptom of cough, blood, dyspnea or pain.

It is very obvious that it would be difficult and almost impossible to establish any criteria of longevity on the basis of correlating symptoms with the onset of the disease, because such evidence would automatically show that some patients, at least in this group of cases, would have lived more than five and ten years without any kind of treatment. Chronic lung infection is undoubtedly coincidental and referable symptoms may subsequently dovetail into the development of a malignant neoplasm.

It seemed necessary, therefore, to establish some uniform basis of calculating or estimating the benefit of radiation treatment by some other method. If the value of irradiation could be considered according to the duration of life from the time of making the diagnosis and instituting treatment, then a fixed routine and a definite standard plan for making comparisons and relative estimations would be greatly simplified. This would be an absolute factor and would permit some practical conclusion in a large group of cases.

In this study, therefore, the value of irradiation was considered according to the life cycle from the time of instituting roentgen treatment, and this was practically

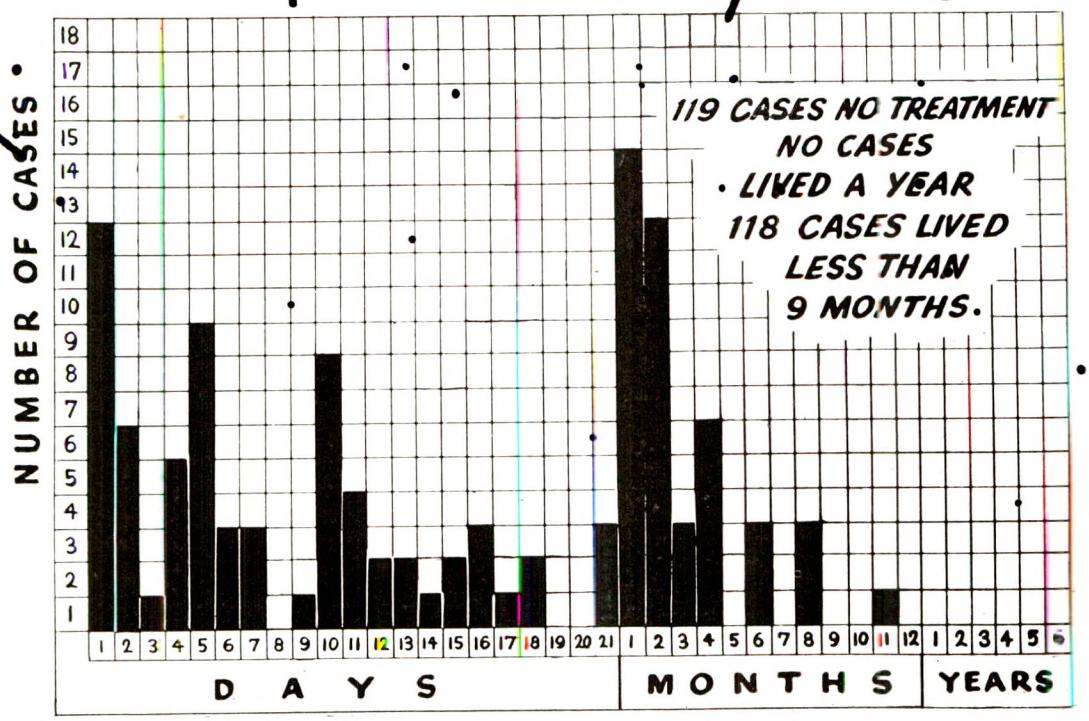


Chart II

always within a few days of making the diagnosis. A histopathologic study was sometimes not obtained until autopsy, but in these cases treatment was instituted on the clinical and roentgenologic evidence. This method definitely establishes a space of time which is accurate for all cases. Obviously the unknown duration and the varied extent of involvement before making the diagnosis precludes any possibility of a mathematical tabulation of the life expectancy of a given case.

The average of these variable factors in a large series of cases probably equalizes the pathologic status of a large number of cases when irradiation is started. The life cycle after irradiation is easily established. Any significant duration of time is obvious and beneficial effects can be reasonably determined. Conclusions cannot be absolute but relative estimations are simplified.

The 119 untreated cases are tabulated in Chart II. No case lived as long as one year after the diagnosis was made; 112, or 94 per cent, lived less than six months.

Some of these patients on admission were too ill for any kind of treatment to be considered. On the other hand, many were comparable with the clinical and physical status of the treated group but received no treatment at a time when they were all regarded as hopeless, before there was any interest in irradiation or before technical experiences with irradiation were developed. It would, therefore, be futile to make direct comparisons with the treated and untreated cases, but there is a relationship which must be given some consideration, and can readily be visualized by comparison of Charts II and III.

Of the 167 treated cases, 18 lived twelve months or more from the time of making the diagnosis (Chart III). Five cases lived two to six years as follows: 2 cases lived two years; 1 lived three years; 1 lived five years and 1 is still alive six years. The patient still alive six years was forty-three years of age when treatment was started. He now has a persistent dry cough, which may be due to irradiation fibrosis or cancer or both. Repeated bronchoscopic examinations have been negative.

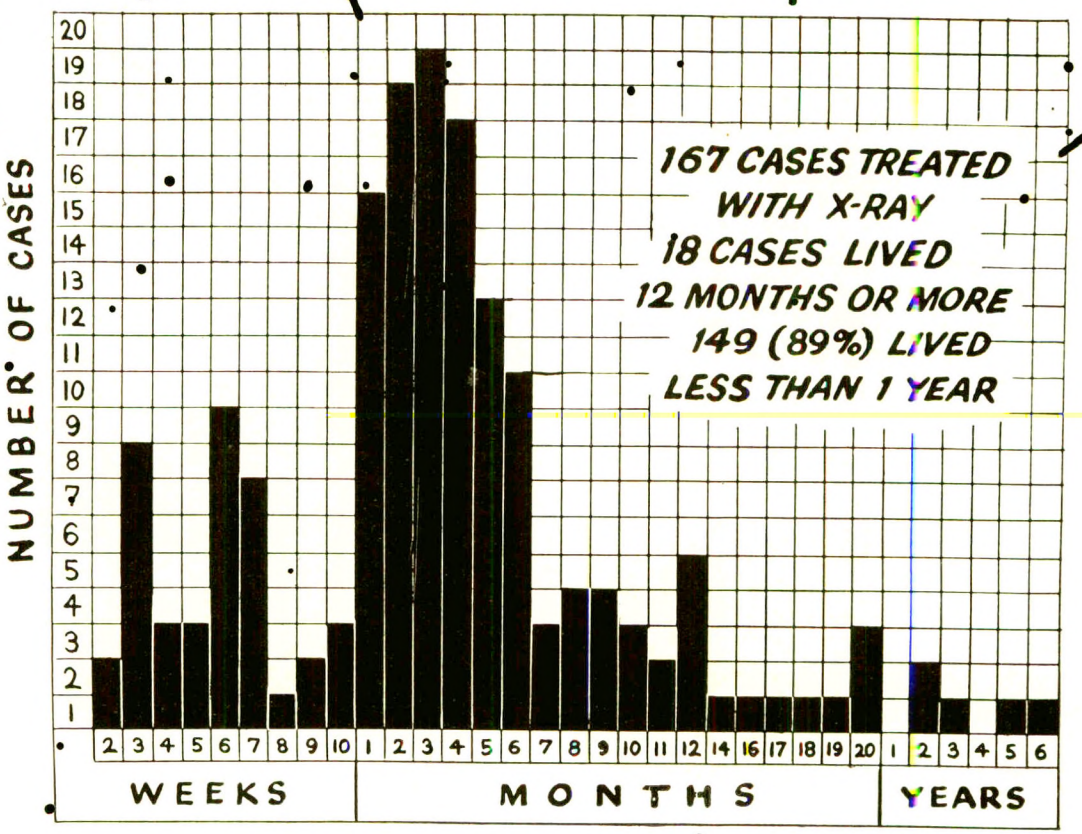


Chart III.

There were 38 cases tabulated as living two to ten weeks, because they were in very poor physical condition and received less than 1,000 tissue roentgens. Of 167 cases, 149, or 89 per cent, lived less than one year; 113, or 67 per cent, lived less than six months.

All of the 5 cases living two years or more received in excess of 6,000 tissue roentgens. The patient still alive six years received 8,400 r. Many cases lived less than one year after total doses of 5,000 to 13,000 r.

There is some suggestion that cases of bronchiogenic cancer may show greater longevity cycles if greater doses of radiation could be instituted at an earlier stage of the disease, before symptoms of weakness and loss of weight develop. This would not be possible in many cases, because these were frequently the presenting symptoms when diagnostic investigations were instituted.

Discussion

The effect of irradiation in primary cancer of the lung is ultimately and almost entirely poor. The cellular character of the tumor seems to bear little relationship to the response to irradiation. Apparent resistance, regardless of the microscopic type of the disease, is very likely due to the anatomic and histopathologic structure of the bed of the disease, accentuated by frequent complications of infections.

The possibility of a malignant neoplasm existing without associated symptoms for a long period of time must be acknowledged. The progress of the disease may be insidious and hopeless stages of development found when the first symptom is manifested.

The majority of cases are advanced and inoperable when the diagnosis is established. Irradiation is the only treatment if the physical condition of the patient permits.

Evaluation of cough, dyspnea, pain, purulent and bloody expectoration, etc., for periods of one year or more are symptoms difficult to correlate with cancer. Such long durations preclude such an inference in all instances. Tuberculosis, silicosis, bronchiectasis, passive pulmonary congestion of cardiac origin, chronic nontuberculous lung infections of upper respiratory origin, and many other inflammatory conditions might explain symptoms of long duration but may likewise be sources of chronic irritation, and the actual predisposing factor in the subsequent development of a malignant neoplasm.

Any effort to evaluate the longevity cycle of a case of bronchiogenic cancer must be futile, unless the diagnosis can be established very soon after the first chest symptom.

Beneficial effects of irradiation according to the duration of life from the time of making the diagnosis offers a crude but reasonably accurate basis of formulating conclusions in a large group of cases, whose status of uncertain duration and extent of involvement possibly average some similar approximation when treatment is instituted.

It is important to consider that none of the untreated cases lived a year as compared with 18 of 169 treated cases living one to six years after instituting treatment. Furthermore, 112 untreated cases, or 94 per cent, lived less than six months, as compared with 113 treated cases, or 67 per cent, who survived less than six months. Eighty-nine per cent of 167 treated cases lived less than one year.

This evidence shows that irradiation may prolong life one to six years in approximately 10 per cent of the cases after instituting treatment.

The advantage is not spectacular when account is taken that approximately 90 per cent of irradiated cases lived less than a year as compared with 99 per cent of the untreated cases living less than nine months.

SUMMARY

1. The prognosis was poor in this series of 286 cases of proved bronchiogenic cancer because of the advanced stage of the disease at which a correct diagnosis was made.

2. Every patient, except the few cases that may be considered as operable, should be given the benefit of every advantage which irradiation can offer and within the limits of skin and physical tolerance.

3. Since no patient who was not irradiated survived longer than a year, and since 18 patients who were irradiated lived one to six years, the value of roentgen therapy as a palliative procedure for bronchiogenic cancer seems unquestionable.

4. Irradiation may prolong life a year or more in approximately 10 per cent of cases, regardless of the microscopic cellular character of the lesion.

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DISCUSSION

DR. U. V. PORTMANN, Cleveland, Ohio. When discussing results of roentgen therapy for bronchiogenic cancer, it is necessary to compare them with other methods of treatment and explain the reasons for successes and failures. When considering either surgical or radiological procedures for malignant tumors, we are confronted with technical and biological limitations due to growth characteristics of tumors which influence anatomical extent of involvement or accessibility.

The first technical difficulty is the diagnosis. The symptoms, physical signs, and roentgenological features of bronchiogenic cancer may simulate many diseases. The onset is insidious, and symptoms obscure; therefore, in many cases extension has taken place to the pleura or mediastinal structures, including the lymph nodes, before a diagnosis is made. Clinical and roentgenological manifestations depend upon the duration, location, extent of involvement by the neoplasm, and degree of obstruction to lung tissue. Therefore in the absence or presence of symptoms or signs that suggest bronchiogenic cancer, it should be considered when a parenchymal shadow is seen in the roentgenogram of an adult lung because a bronchiogenic tumor seldom can be visualized except by special techniques but the parenchymal shadow is made principally by reactionary or secondary pneumonitis.

Since a positive diagnosis of bronchiogenic cancer cannot be made from clinical or roentgenological characteristics, other procedures must be resorted to. No doubt bronchoscopic visualization and biopsy are most valuable and successful in about 65 per cent of cases, because these tumors originate in or extend into main bronchi and are accessible. Aspiration biopsy under fluoroscopic control is sometimes useful when tumors are inaccessible bronchoscopically. Microscopic examination of aspirated pleural fluid may be helpful if tumor cells are found, but if they are not demonstrable the method is valueless. Finally, a diagnosis may be established by thoracotomy or autopsy, the latter too frequently following immediately after the former, and neither helping much in treatment.

In discussing the results of roentgen therapy for bronchiogenic cancer, we should also consider its indications and limitations with surgical procedures.

Surgeons have said that thoracotomy is indicated (1) when a diagnosis has been established but "operability" is questionable; (2) when primary cancer is suspected, but not proved pathologically; (3) for cases with proved bronchiogenic cancer but without clinical manifestations of metastases and in spite of roentgenological evidence of possible mediastinal involvement which may be only lymph nodes enlarged by inflammation; (4) even with complete atelectasis of a lung which may be caused by edema secondary to cancer or other type of tumor such as a polyp in a bronchus. Also that pneumonectomy is indicated even if there is direct extension into the chest wall which may be excised, but this condition decreases the possibility of cure because metastasis may have occurred through intercostal lymphatics.

The contraindications to thoracotomy are said to be (1) obvious distant metastases, (2) cancer located at the carina or in the trachea, (3) presence of Horner's syndrome indicating mediastinal involvement of sympathetics.

According to these indications and contraindications most patients with bronchiogenic cancer are "operable." But results reported by courageous surgeons of very few cases surviving five years are not convincing that many are "surgically curable" or even benefited because of technical difficulties due to extent of involvement. However, many patients are treated by roentgen therapy for bronchiogenic cancer with and without positive diagnosis because of contraindications to operation.

There are biological and physical difficulties

that confront radiologists in the treatment of bronchiogenic cancer.

Biological difficulties are due to the variability of growth characteristics of these neoplasms. They originate from epithelial cells in basal layers of the bronchi or bronchioles, some from squamous cells in the mucosa, others from secretory cells in mucous glands. But no matter where they originate the degree of differentiation of parent cells determines the growth characteristics which are responsible for tumors of different histopathological types that may be predominantly squamous cell, adenocarcinoma or round, spindle or oat cell carcinoma. However, bronchiogenic cancer is not one of these histopathological types uniformly throughout but the morphology varies in many areas although one type may predominate. Likewise the degree of differentiation of the neoplastic cells also varies considerably in different tumors and in many areas of any tumor no matter what the predominant histopathological type.

These biological characteristics explain the physical difficulties of roentgen therapy.

We know that undifferentiated neoplastic cells may be radiosensitive relative to those that are highly differentiated and radioresistant and that most epithelial neoplastic cells have the latter characteristic. Also that neoplastic epithelial cells of a bronchiogenic cancer of any histopathological type vary in degrees of differentiation both qualitatively and quantitatively in many areas. Therefore some cells or parts of a neoplasm may be radiosensitive but the majority are radioresistant. We have learned that few cancers in any location are so radiosensitive that they can be destroyed by as little as 5,000 tissue roentgens and most require twice as much. But at present it is impossible to administer 10,000 tissue roentgens with impunity to areas involved by bronchiogenic cancer. Also we probably do not administer so much as calculated from dosage charts because aerated lung surrounding a tumor is not such a good scattering medium as solid materials used to measure dosage in physics laboratories. Therefore though some neoplastic cells in bronchiogenic cancers may be destroyed by roentgen therapy with quantities that can be administered, it is biologically and physically impossible to destroy all of them.

If this be conceded, we may ask why and when treatment is indicated and what results or benefits may be expected. Unfortunately some clinicians judge benefits of therapeutic procedures principally from statistical evidences that the percentage of survivals for a specified time is higher in comparison with other methods or no treatment. But not all benefits can be calculated mathematically. Although radiologists may be unable to prove statistically that roentgen irradiation has cured patients with bronchiogenic cancer and they are reluctant even to maintain that lives have been prolonged, still there is adequate objective and subjective evidence that benefits have been derived by extension of economic usefulness and palliation.

No doubt irradiation destroys some neoplastic cells of bronchiogenic cancer and diminishes the vascularity by effects on capillaries thus reducing the mass which may relieve bronchial obstruction. But, in my opinion the greatest benefits are derived from effects of irradiation upon lung tissues and enlarged lymph nodes not necessarily involved by cancer but affected secondarily by concomitant infection and inflammation which are responsible for atelectasis or obstructive pneumonitis. This condition in the lung will be affected by irradiation in the same way that inflammation in other tissues may be reduced in intensity and extent. When this happens the aeration of the lung will be increased by relief of bronchial obstruction manifest by alleviation of cough, dyspnea, expectoration, hemoptysis and pain, which patients appreciate.

Subjective improvement after treatment also may be demonstrable objectively in roentgenograms which often show increased aeration of the lung, diminution in the density and extent of pneumonitis and of pleural effusion. But this apparent improvement should not be misleading. It may not mean so much reduction in extent of involvement from the neoplasm as diminution of the obstruction pneumonitis.

These subjective and objective benefits suggest that roentgen therapy could be given for bronchiogenic cancer before surgical procedures are instituted to reduce inflammation and obstructive pneumonitis so that a better conception might be gained of the actual extent of involvement, the condition of the patient improved and operation facilitated.

ROENTGEN STUDY OF LYMPHOGRANULOMA VENEREUM*

REPORT OF TWENTY-FOUR CASES

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WITHIN the last few years lymphogranuloma venereum has received increasing medical study. The disease was fully described in 1890.[7] In 1901 the frequency of rectal involvement, as a part of the syndrome, was noted by Marion and Candy.[6] There was continuous research in the field with an important paper by Durand, Nicolas and Favre[2] appearing in 1913. The clinical aspects were well explored by 1925 when Frei[4] offered a diagnostic method of establishing the disease. There has been little work done in the roentgenological field. Rendich and Poppel[8] in 1939, and Steinert[9] in 1940 made some roentgen studies. Buckstein[1] also reported a few roentgen studies of the disease in his book which appeared in 1940.† It seems of definite value, however, to add the following cases, with a detailed study of the roentgen findings, to the literature on the subject.

Lymphogranuloma venereum appears first in males as a minute erosion or papule on the penis or prepuce; in females the infection is carried not to the inguinal glands but to the wall of the rectum causing a great infiltration of the outer wall of the part of the colon that presses upon the lumen.[5] The symptoms of the disease, tenesmus, constipation and mucous and bloody stools, are caused by rectal strictures above the anal orifice and ulcerating and granulating tissue covering the entire rectum. Roentgen studies show that in cases of lymphogranuloma venereum there is a destruction of the mucosa, rectal strictures, distention of the rectal pouch, single and multiple perirectal and perisigmoid sinus formation and fistulous tracts. (It may be noted that the formations are originally sinus but may become fistulous.)

Usually the roentgen picture of the disease is clear and of diagnostic value in identifying the conditions we have just mentioned. The cases summarized in Table 1 attest to this method of diagnosis. It can be seen that some of the cases were negative by the Frei test; others were operated on because of mistaken diagnosis but all of the cases showed marked evidence of the disease when they were examined roentgenologically.

REPORT OF CASES

For the purpose of illustrating more fully the type of lesion seen in roentgen studies of these cases of lymphogranuloma venereum we present the following cases.

CASE XII. R. D., male, Negro, aged twenty-one, was admitted to the hospital February 24, 1938. He gave a history of tenesmus and loose stools dating back a year. The physical examination revealed palpable masses in the rectum. The proctoscopic examination showed a thickening of the rectal mucosa with ulcerations on the left lateral wall 2 in. above the sphincter. The Wassermann reaction was negative. The Frei test was positive. The biopsy showed an inflammatory process. The roentgen examination showed a uniform narrowing of the rectum (Fig. 1).

CASE XIX. L. A., female, Negro, aged twenty-eight, was admitted to the hospital November 16, 1939. She complained of hemorrhoids. This condition began in 1936 and she was operated on for it at that time. The physical examination, at the time of her admission to the hospital showed a stricture 3 in. above the external sphincter and a thickening of the rectovaginal septum. The laboratory report showed a nega-

† An excellent paper "Venereal Lymphogranulomatous Rectal Stricture" by Helper and Szilagyi appeared in this JOURNAL, August, 1942, some months after this article was written.

* From the Roentgen Departments of the New York City Hospital and the New York Cancer Institute.

tive Wassermann reaction and a positive Frei test. Roentgen findings disclosed an extensive rectal stricture with multiple sinus formations in the perianal region (Fig. 2).

CASE XXIV. K. W., male, white, aged forty, was admitted to the hospital November 12, 1941. Seven years previous to his admission he had had an enlarged lymph node in the right groin which was incised. Then, he developed a diarrhea with blood and mucus. He received no satisfactory treatment until 1940 when he was given sulfanilamide and injection treatments. At the time of admission to the hospital he complained of diarrhea, painful defecation and constipation necessitating the use of enemas. The physical examination showed an inguinal scar and a rectal stricture. Laboratory examination showed a 4 plus Wassermann reaction. The Frei test was unsatisfactory. Roentgen examination revealed an extensive narrowing of the rectum with numerous sinus formations and retention in the sinuses after evacuation (Fig. 3).



FIG. 1. Case XII. Stricture of the entire rectum with tubular narrowing secondary to lymphogranuloma venereum.

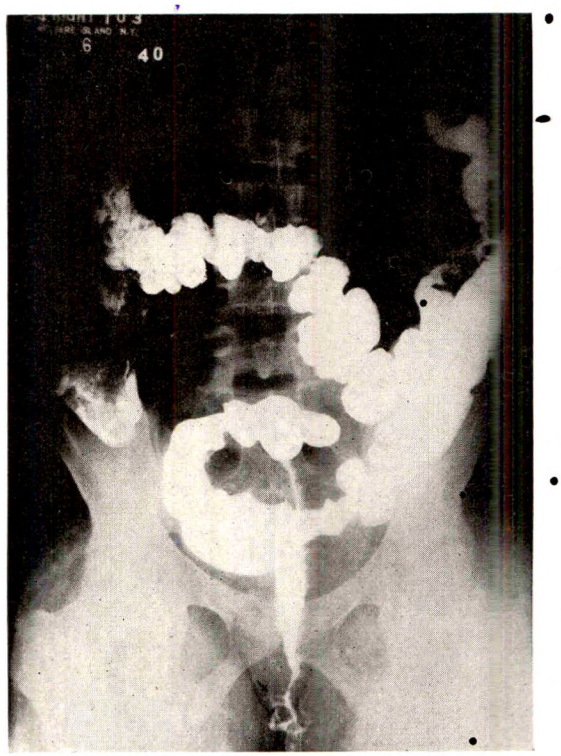


FIG. 2. Case XIX. Extensive rectosigmoid narrowing with perianal sinuses.

CASE XXIII. N. B., female, Negro, aged forty-three, was admitted to the hospital July 10, 1941. She gave a history of diarrhea associated with abdominal pain without blood or mucus. This condition was present for five months previous to her admission to the hospital. The physical examination was negative, except the rectal examination which showed a papillomatous nodule 1.5 in. in with associated stricture. The laboratory report stated that the Wassermann reaction was negative and the Frei test positive. Roentgen examination displayed a distention of the rectal pouch and a pronounced rectosigmoid stricture with a long irregular sinus (Fig. 4).

CASE XV. V. S., female, Negro, aged forty-four, was admitted to the hospital July 30, 1937. She gave a history of having rectal bleeding and abdominal pain for the past two years. A physical examination showed no rigidity nor masses in the abdomen. The rectal examination revealed many rectal tabs above the anal orifice. Three inches above the external sphincter was an annular constriction. Proctoscopic exam ina-

TABLE I

Case No.	Date	Name	Age	Sex	Race	History	Physical Examination*	Laboratory Findings	Pathological Findings	Roentgen Findings
I	11-12-31	W.H.	48	M	W	1 yr. history of tenesmus, rectal bleeding and constipation; hemorrhoidectomy; later colostomy	Rectal stricture with multiple papillomatous lesions	Wass. + Frei	Biopsy—chronic inflammation	Distended rectosigmoid pouch; rectal stricture
II	6-18-32	C.V.	28	F	N	Painful defecation for 3 mo.; pencil stools; loss of weight	Rectal stricture	Wass. − Frei +	Biopsy—inflammatory tissue	Extensive inflammatory rectal stricture
III	6-20-32	A.H.	34	F	N	Fistula in ano, 1925; rectal stricture with increasing constipation, 1926; perirectal abscesses, 1929.	Rectal stricture	Wass. + Frei +	Autopsy—marked perirectal fibrosis and obstruction	Narrowing of rectosigmoid
IV	2-8-34	G.Y.	50	M	W	Increasing constipation, diarrhea and rectal bleeding; treated with roentgen therapy for rectal cancer without biopsy; re-admitted 2 yr. later with explosive diarrhea	Rectal stricture 3 in. above anal orifice	Wass. − Frei −	Biopsy—chronic granulomatous condition	Narrowing, irregularity and fixation of rectosigmoid
V	8-9-35	C.A.	55	M	N	Increasing painful defecation 5 yr. with rectal bleeding	Rectal stricture	Wass. Frei +	Biopsy—extensive inflammatory process	Sausage-shaped deformtiy of rectosigmoid
VI	1-3-36	J.D.	64	F	N	20 yr. history of constipation, tenesmus and stringy stools; two operations with temporary relief	Rectal stricture above anal orifice	Wass. − Frei +	Biopsy—inflammatory productive changes	Narrowing of rectosigmoid
VII	9-2-36	H.M.	76	M	W	History of rectal condition since 1934 with mucoid discharge the past 3 mo.	Rectal stricture at anal orifice with adjacent area of induration	Wass. − Frei +		Rectal narrowing
VIII	6-2-37	C.S.	45	F	N	Increasing constipation and diarrhea since 1915. Rectal dilatations, 1915–1921	Rectal stricture	Wass. Frei +		Narrowing in rectum and lower sigmoid
IX	8-25-37	F.C.	42	F	N	4 yr. history of increasing constipation associated with rectal bleeding	Rectal stricture	Wass. − Frei +		Rectosigmoid stricture
X	10-26-37	P.Z.	35	M	W	25 yr. history of progressive constipation and associated diarrhea. Recent colostomy	Rectal stricture	Wass. + Frei +		Narrowing in rectum to rectosigmoid
XI	1-15-38	P.J.	27	F	N	Rectal pain and discharge since June, 1937; radium treatment, relief for 1 mo., followed by colostomy	Rectal ulceration, necrosis of buttock, coccyx to vulva	Wass. − Frei +	Autopsy—chronic productive inflammation in perirectal tissue	Narrowing deformity of entire rectosigmoid
XII	2-24-38	R.D.	21	M	N	1 yr. history of tenesmus with loose stools	Palpable masses	Wass. − Frei +	Biopsy—inflammatory process	Uniform narrowing of rectum
XIII	4-12-38	B.G.	34	M	W	2 yr. history of rectal pain; operation; 1 yr. of later developed inguinal masses, swelling of scrotum, rectal discharge	Rectal stricture 2 in. above anal orifice	Wass. + Frei +		Dilatation of rectosigmoid pouch and rectal stricture
XIV	5-2-38	P.K.	59	M	W	1 yr. history of abdominal pain and progressive constipation; colostomy performed	Rectal stricture 1 in. above anal orifice	Wass. − Frei +	Biopsy—inflammatory tissue	Irregularity and narrowing of rectum
XV	7-19-38	V.S.	44	F	N	2 yr. history of rectal bleeding associated with abdominal pain	Annular constriction	Wass. − Frei −	Biopsy—chronic granulation tissue	Marked narrowing of rectum, sigmoid and distal descendens. Small sinuses
XVI	1-6-39	B.A.	39	F	N	Constipation 3 yr. associated with difficulty in defecation	Rectal stricture; vaginal fistulae	Wass. − Frei	Biopsy—chronic granulation tissue	Rectal stenosis
XVII	7-22-39	B.R.	42	F	N	15 yr. history of recurrent perineal abscesses, rectal stricture and rectovaginal fistula. Two opertions 9 and 5 yr. previously, temporary relief; June, 1930 cecostomy for cancer of rectum	Rectal stricture	Wass. − Frei +	Biopsy—chronic granulation tissue	Smooth narrowing of rectosigmoid suggesting inflammatory stricture; nonmalignant
XVIII	9-4-39	P.C.	39	F	N	Gradual abdominal distention associated with pain in lower abdomen	Rectal stricture 2 in. above anal margin	Wass. − Frei +		Tubular narrowing of rectum to 5 cm. beyond rectosigmoid junction
XIX	11-10-39	L.A.	28	F	N	Hemorrhoids since 1936.	Rectal stricture; thickening of rectovaginal septum	Wass. − Frei +		Extensive rectal stricture with perianal sinuses
XX	1-14-41	D.T.	18	F	N	2 mo. history of rectal bleeding, diarrhea and painful defecation	Rectal stricture	Wass. − Frei +		Narrowing of rectum and sigmoid
XXI	1-22-41	B.P.	29	F	N	Increasing rectal pain and difficulty in defecation	Rectal stricture	Wass. + Frei	Biopsy—chronic granulation tissue	Irregularity of anal canal with smooth stricture of distal two-thirds of rectum
XXII	6-7-41	R.R.	39	M	N	Increasing constipation with associated abdominal cramps	Rectal stricture	Wass. Frei +	Biopsy—unsatisfactory	Concentric narrowing of rectum
XXIII	7-10-41	N.B.	43	F	N	5 mo. history of diarrhea associated with abdominal pain	Rectal stricture; papillomatous nodules	Wass. − Frei +		Rectosigmoid stricture; large sinus tract
XXIV	11-12-41	K.W.	40	M	W	7 yr. history of constipation, diarrhea, bloody stools with mucus necessitating frequent enemas	Rectal stricture	Wass. + Frei		Rectal narrowing with numerous retention sinuses

* All males had inguinal scars or palpable glands.

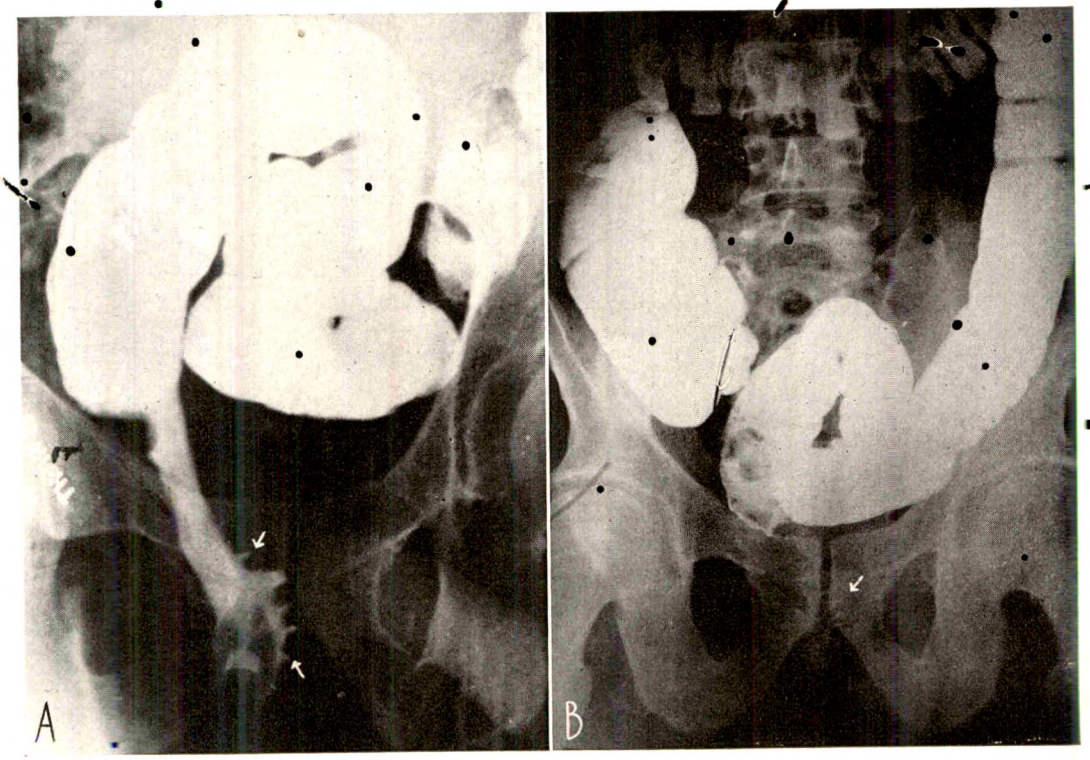


FIG. 3. Case XXIV. *A*, anorectal stricture with tubular narrowing and multiple retention sinuses in the perianal and perirectal tissues. *B*, after evacuation showing the retention in the colon proximally, also perianal and perirectal sinuses.

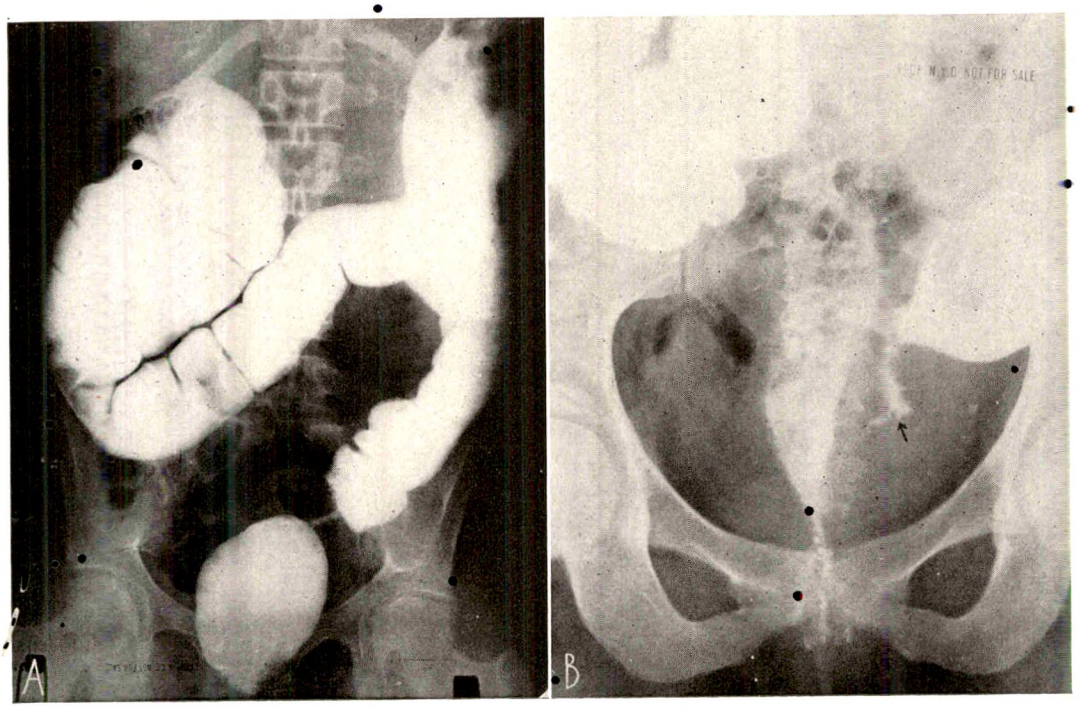


FIG. 4. Case XXIII. *A*, annular stricture of the rectosigmoid secondary to lymphogranuloma venereum simulating cancer. *B*, after evacuation; note the large sinus tract. This tract is very unusual with cancer.

tion showed a marked narrowing of the anal canal extending for 10 cm. On examination it proved to be a benign stricture. The laboratory report stated that the Wassermann and Frei tests were negative. A biopsy showed chronic granulation tissue. Roentgen examination showed a marked tubular narrowing of the rectum and sigmoid and distal descendens with an absence of normal markings. There were fine sinuses extending laterally[1] (Fig. 5).

A study of these cases indicates the various forms that lymphogranuloma venereum of the colon assumes and which can be noted in roentgen studies. However, it is important from the diagnostic angle to point out that these cases of lymphogranuloma venereum have occasionally been mistaken for cancer. Evidently, the duration of the disease is the first differentiation. Cases of lymphogranuloma venereum usually run a chronic course of years, even twenty or more. The history of cases of cancer is generally two or three years. About 75 per cent of cases of rectal cancer have the lesion on the anterior wall.[3] In lymphogranuloma venereum, although the lesions may be localized, more often they circle the wall. Lesions of lymphogranuloma venereum begin in the anal region while cancer tends to develop in the rectum. The cases shown in Figures 6, 7 and 8 illustrate these points of differentiation.

In addition to the difference in the duration and the sites of these two diseases, cancer may be identified since its lesions may cause circumscribed elevations of the mucosa with abrupt terminations and these lesions are associated with ulcerations. Cancer, on roentgen examination, shows an irregular shadow protruding into the lumen. Lymphogranuloma venereum shows, by the same method, more often a regular shadow. The exception is stenosing scirrhous carcinoma which tends to infiltrate and circle the wall and shows a

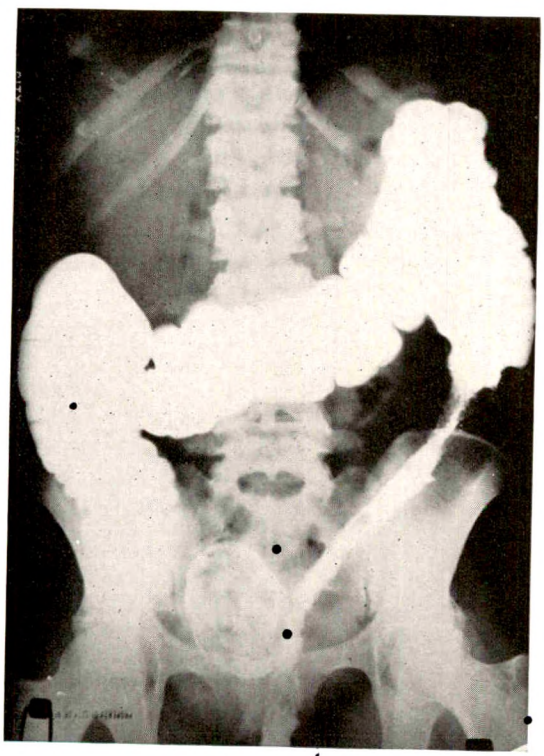


FIG. 5. Case XV. Extensive tubular narrowing involving the rectum, sigmoid and part of the descendens with occasional fine sinuses.



FIG. 6. Malignant stricture of rectal ampulla. Lymphogranuloma can be excluded since the rectum below the stricture, down to the anus, has normal mucosal folds.

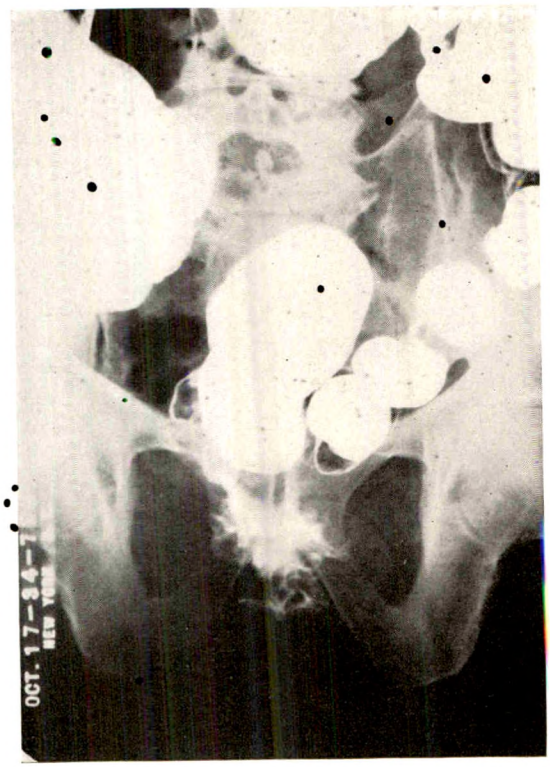


FIG. 7. Cancer of the lower rectum simulating lymphogranuloma venereum as a result of its location. Note the absence of tubular narrowing and fungating luminal deformity.

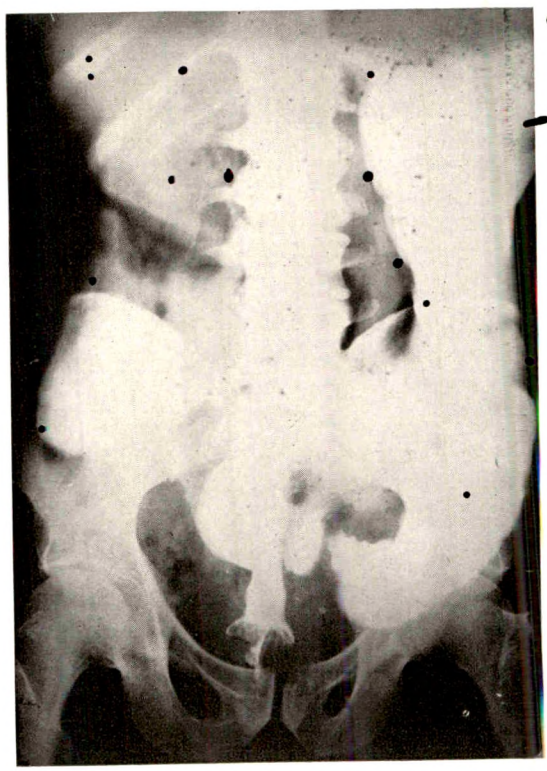


FIG. 8. Cancer of the proximal rectum with annular narrowing. Broadening mucosal pattern distally fading into a normal mucosal fold at the anorectal junction.

smooth shadow on roentgen examination. Ruptured multiple diverticula associated with cancer is the other exception in considering the differential diagnosis.

SUMMARY

Lymphogranuloma venereum of the colon, as studied in 24 cases, tends to develop certain characteristics which can be shown definitely by roentgen examination. These characteristics are destruction of the mucosa, rectal stricture, distention of the rectal pouch, single and multiple perirectal and perisigmoid sinus formation and fistulous tracts. The condition is sometimes mistakenly diagnosed without a roentgen examination. It therefore seems advantageous to use the roentgen ray as an aid in establishing the diagnosis and extent of this disease.

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DATA ON THE ATTENUATION OF NARROW AND BROAD BEAMS OF 1,000 KILOVOLT (PEAK) ROENTGEN RAYS BY LEAD, CONCRETE AND WATER*

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THE protection of operating personnel from supervoltage radiation requires the construction of relatively costly and inconveniently bulky barrier walls. For radiations corresponding to 600 kilovolts, constant potential, and below, the amount of lead or other material required is relatively small. It can be satisfactorily added to the walls of an existing structure, and applied according to authoritative recommendations now in existence.[1,7] But for radiations corresponding to voltages in the neighborhood of 1,000 kv. (peak) and above, the great weight and thickness of the necessary walls introduce serious architectural problems. Special walls are required, and often special buildings; and the space occupied by the walls often equals the useful treatment room space.

In the supervoltage region, therefore, it becomes most profitable to choose with care the most suitable barrier materials and to calculate precisely the necessary and sufficient thickness. Unfortunately much less experimental data have been available for this purpose than in the case of softer radiations. Theoretical treatment must take into account radiation that is scattered by walls which are struck by rather wide roentgen-ray beams. In the region of 1,000 kv. (peak) the problem is complicated by the great thickness of the walls required and by the fact that the mechanism of absorption is still dependent, to some extent, upon the nature of the absorbing material used.[2]

Although a number of other special materials have been proposed, protective barriers are generally constructed of lead or concrete, or the two combined. The attenuation due to distance is called upon whenever possible in order to reduce the amount of lead or concrete required. In general, in supervoltage therapy, the use of lead is restricted to regions close to the tube anode, and concrete to walls and floors distant from it.

Concrete barriers are usually subjected to broad beams of radiation. They must reduce the intensity of these broad beams to below the tolerance level. Concrete is a relatively light material, composed of elements of low atomic weight, and consequently can be expected to scatter very high voltage radiations somewhat as water does.[2] The thickness of concrete required must at present be calculated from absorption data, with allowances being made for the scattering of radiation out of the broad beams penetrating the concrete. The importance of this scattering factor is demonstrated by the experiment described at the end of this paper.

With the aid of the supervoltage therapy equipment at Memorial Hospital, it was possible to measure the attenuation of narrow beams of 1,000 kv. (peak) roentgen rays in very thick blocks of lead, concrete, and water. It was found possible to study one special case of the general problem of the penetration of broad beams through concrete.

EQUIPMENT

The roentgen-ray equipment used[3] consisted of a cascade type tube housed in a pressure tank together with its transformer. A 180 cycle, alternating potential, with peaks of 1,000 kv. could be applied to the tube. Unfortunately, this potential could

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not be measured during the experiment but depended upon the calibration at the factory. It was, however, readily reproducible and could be monitored by the "charging current" drawn by the transformer. The tube current at full voltage was maintained at 3 milliamperes.

The anode of this tube is shielded by a housing of lead, steel, and mercury, so designed that a beam of radiation may be directed at any angle between vertically downward and the horizontal.[4] For the following experiments only the downward direction was used, the radiation being in the same direction as that of the cathode rays. The radiation passed through the tungsten target, the water jacket, and then through about 2 mm. of mercury. The approximate inherent tube filter is stated in Figure 2.

The focal spot was measured by means of pin hole photographs and was about 2 cm. in diameter.

NARROW BEAM ABSORPTION MEASUREMENTS

In the first instance, and following the procedure generally used at lower voltages,[1] the true absorptions of lead, concrete, and water were determined by using a narrow beam of radiation. In this manner the behavior of concrete as an absorber could be compared with that of a lighter material (water) as well as that of a heavier material (lead).

Figure 1 shows the experimental setup in cross section. A hole in the floor of the treatment room, directly below the target, permitted a narrow collimated beam to be projected into the well shielded room below. The floor of the treatment room is made of concrete and is 3 feet thick. Very little leakage through this floor was detected.

The intensity of radiation was measured by thick walled thimble chambers connected to an electrometer tube network and current compensator. The ionization chamber could be placed at a great distance from the filter, as shown in Figure 1.

The limiting diaphragm, 10 cm. thick, shown in Figure 1, permitted a beam to pass through the hole in the floor without grazing the latter. When lead fore-filters were used they were placed just above the limiting diaphragm. The heavy concrete slabs, as well as the tank of water, were placed on the floor of the treatment room over the hole.

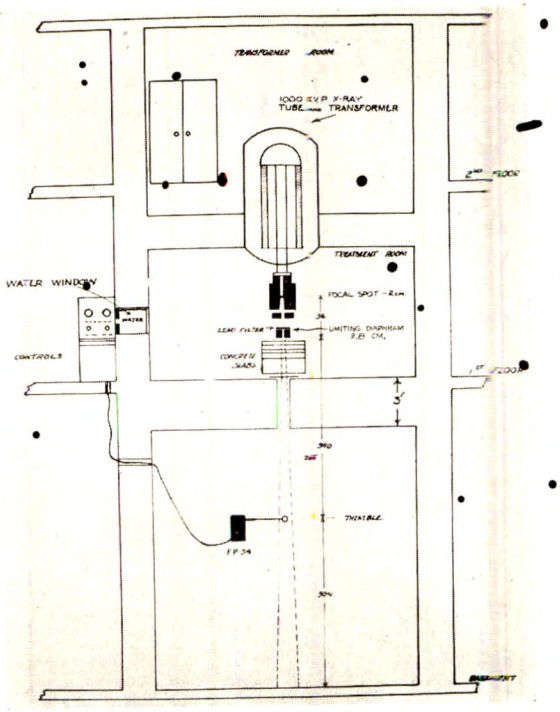


FIG. 1. Technique of determining the absorption of narrow beams by concrete, water, and lead. A cross section of the transformer and tube is shown, as well as the positions of filters and measuring instruments. Distances in centimeters.

CONCRETE AND WATER ABSORBERS

The concrete blocks were made of Portland cement, sand and small gravel (up to about 8 mm. in diameter), in the ratio of about 1:2:2. It was wet-cast into wooden frames, 2 feet square and with various thicknesses. A slight tamping, corresponding to conventional builder's practice, was given the wet material and it was then set aside to dry indoors for several months. The blocks were then weighed and measured. The specific gravity of the seasoned blocks averaged 2.2 and varied among the samples from 2.13 to 2.23.

Two blocks were made of 2 inch thickness, and one each of 4, 6 and 10 inch thickness. In this way the same nominal absorber thickness could be obtained from several combinations of the blocks. The transmissions through all possible combinations of the blocks, as well as through different regions of individual blocks and combinations, were determined and practically no inhomogeneity was discovered. The density of the concrete was satisfactorily uniform.

The water absorber consisted of a tank having a paraffined paper bottom. It was placed over the hole in the treatment room floor. It could be filled to a depth of about 60 cm. and the attenuation due to any chosen thickness of water determined.

ABSORPTION CURVES

Figure 2 shows the absorption of the narrow beam in lead and concrete as determined with the technique described above, the initial filtration being that inherent in the tube head. (See value in Figure 2.)

Figure 3 compares the attenuations of the narrow beam by water, concrete and lead, with several lead thicknesses used as fore-filters. The concrete and water curves are almost straight lines on the semilog plot, within the experimental error. It is evident that concrete, like water, adds very little to the hardness of the transmitted beam, whereas lead continues to absorb 1,000 kv. (peak) radiation selectively even after as much as 40 mm. of lead are penetrated.

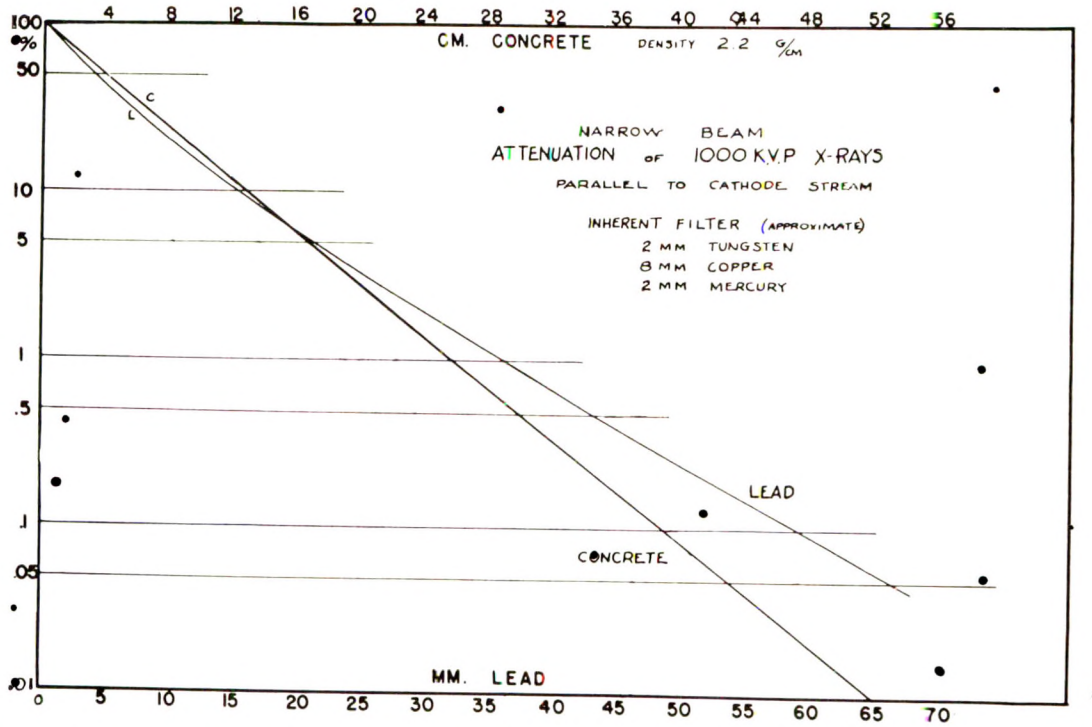


FIG. 2. Attenuation of narrow beams by lead and by concrete determined by technique shown in Figure 1.

If desired, the "lead equivalent" of any thickness of concrete up to 50 cm. can be obtained from Figure 2. The "lead equivalent" is defined as[1] the thickness of lead which will have the same true absorption for the given roentgen-ray beam as the protective material in question. A comparison of the "lead equivalents" for 1,000 kv. (peak) radiation and those for radium and lower voltage roentgen rays are given in Figure 4.

The mass and electronic absorption co-

efficients in lead, concrete and water from the data of Figures 2 and 3 are given in Table 1. The electronic scattering coefficients are calculated on the assumption that the effective atomic weight of concrete is not far from that of silica. The accuracy implied by the data in Table 1 is, of course, presumptuous, considering the assumptions made, but three significant figures are shown to illustrate the trend in the coefficients as the beam of radiation is progressively hardened by the addition of lead fore-filters. It is evident that 1,000 kv. (peak) radiation is absorbed in concrete in the same manner as it is absorbed in water, namely through the Compton process.

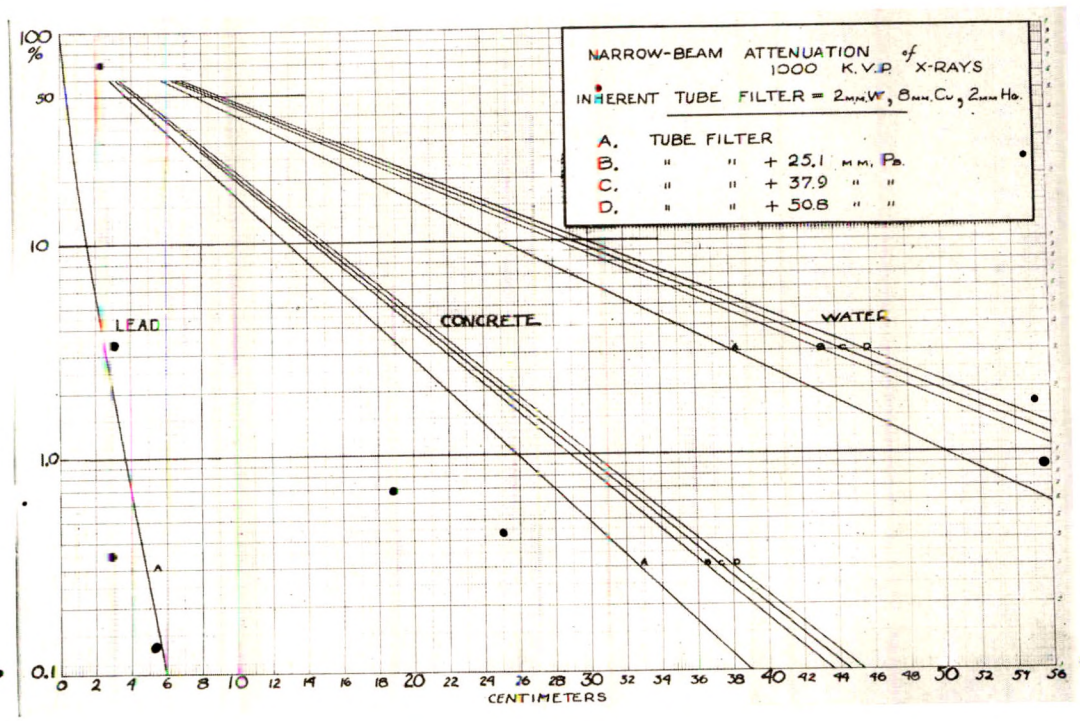


FIG. 3. Absorption of the narrow beam (shown in Fig. 1) by lead, concrete, and water, showing the effect of addition of extra lead fore-filters.

PENETRATION OF BROAD BEAMS

The experimental work of Kaye, Bell and Binks[5] illustrates very clearly the magnitude of the scattered component of a broad beam of gamma rays penetrating a protective barrier. They compared the penetration of a brick wall by gamma rays of narrow and very broad beams. From their work it can be concluded that, if the thickness of a brick wall were to be determined from the narrow beam attenuation coefficients corresponding to gamma radiation, at least two and one-half additional half-value layers of brick would be required to make the wall a safe barricade. This amounts to thickening the wall about 40 per cent. The hardness of radiation used by the above authors was equivalent to about 1,500 kv. monochromatic.

It was thought worth while to determine experimentally the order of magnitude of the effect of broadening a beam of 1,000 kv. (peak) radiation penetrating a thick block of concrete. Unfortunately a full sized wall could not be set up at this laboratory and so only the special case of a moderately broad beam, of a size often found in therapy, with the ionization chamber close to the scatterer could be tested.

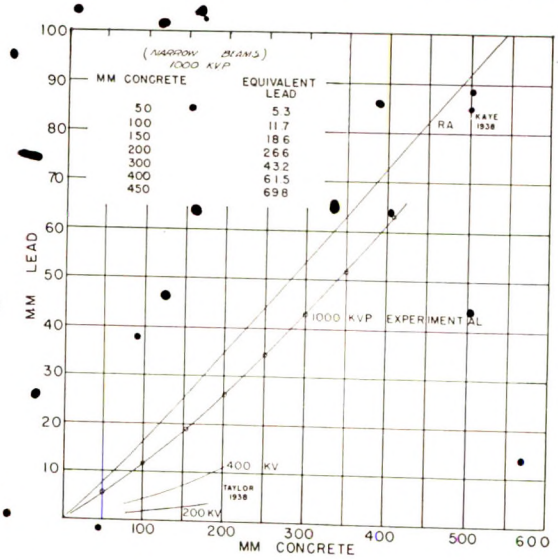


FIG. 4. Equivalence of lead and concrete as absorbers of narrow beams of 1,000 kv. (peak) roentgen rays, as well as values for radium given by Kaye[5] and for lower voltage roentgen rays given by Taylor.[7]

Figure 5 shows the experimental setup. A concrete block was chosen of such a thickness (14 inches) and placed at such a distance from the target that approximately the accepted tolerance dose was transmitted. The concrete block was suspended in mid-air by means of a cradle held by chain tackle. The intensity of radiation at 50 cm. from the target was 100 r per minute, our usual therapy intensity. The filter was that inherent in the tube.

The thimble chamber, having about 4 mm. wall thickness, was placed at a chosen distance (marked x in Figure 5) from the lower face of the concrete block. The concrete was first exposed to a beam 30 cm. in diameter and the transmitted intensity determined. Then, without varying any other condition, the beam diameter was reduced to 10 cm. by inserting a constricting diaphragm at B, see Figure 5. This procedure was repeated for several values of x.

For each value of x the ratio of intensity transmitted by the wide beam over that transmitted by the narrower one was determined and these are given in Table II.

Since some radiation necessarily struck the floor below the suspended blocks and was partly scattered back, this error had to be accounted for. This was done by displacing the thimble chamber horizontally so that it was out of the direct beam, and at the same time shielding the thimble with a block of lead from that radiation scattered from the suspended concrete, but not from that coming from the floor and lower walls. This procedure yielded only a first order correction, but the results showed that this correction factor was fortunately small. The ratios corrected in this manner are shown in the third column of Table II.

Even the 10 cm. diameter beam cannot be considered truly narrow in this instance, especially when the value of x was small. Some scattered radiation would reach the chamber when it was close to the concrete.

TABLE I

ATTENUATION OF NARROW BEAMS OF 1,000 KV. (PEAK) ROENTGEN RAYS

Absorption Coefficients from Slopes of Experimental Curves in Figure 3

Fore-filter	Concrete			Water			Lead		
Lead mm.	μ cm.$^{-1}$	μ/ρ	e^{σ}	μ cm.$^{-1}$	μ/ρ	e^{σ}	μ cm.$^{-1}$	μ/ρ	e^{σ}
Tube	0.177	0.0805	2.71×10^{-25}	0.0920	0.0920	2.73×10^{-25}	1.88	0.166	6.91×10^{-25}
+25.1	0.158	0.0718	2.41	0.0808	0.0808	2.40	1.11	0.098	4.06
+37.9	0.155	0.0705	2.37	0.0789	0.0789	2.34	1.01	0.089	3.72
+50.8	0.152	0.0691	2.33	0.0772	0.0772	2.29	1.00	0.088	3.68

μ = absorption coefficient per centimeter.
μ/ρ = mass absorption coefficient.
e^{σ} = electronic absorption coefficient.

Its magnitude could be determined by using the data of the experiment shown in Figure 1 where the concrete absorber was at a great distance from the thimble chamber. The ratio of measured intensity due to the wide beam over the intensity calculated from the narrow beam attenuation curves was determined. These values are shown in Table III.

CONCLUSIONS

The data in Table III indicate the order of magnitude of the error which might arise when one calculates the thickness of a concrete barrier wall by using nothing more than the ordinary narrow beam attenuation coefficients pertaining to concrete and to this potential. At least two additional half-value layers of concrete would be needed to compensate for scattering. This means that an additional thickness of 8 cm. of concrete must be added to the value calculated on narrow beam data.

The results of the experiment described above and illustrated in Figure 4 answer,

TABLE II

Distance of the thimble from the concrete block in centimeters	Ratio of intensity due to 30 cm. beam over that due to 10 cm. beam	
	Measured	Corrected for floor scattering
6.5	2.5	2.4
17.5	2.3	2.1
35.0	1.8	1.6
122.0	1.4	1.4

TABLE III

Distance of the thimble from the concrete block in centimeters	Ratio of the corrected intensity due to the 30 cm. beam over the intensity calculated from narrow beam attenuation curves
6.5	3.3
17.5	2.4
35.0	1.6
122.0	1.4

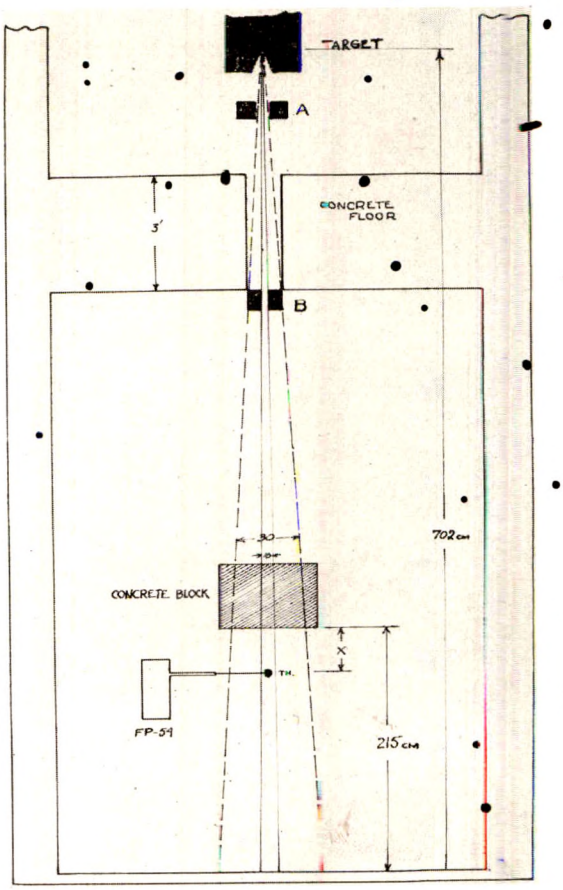


FIG. 5. Method of studying the effect of the width of a beam on the penetration of a thick concrete block.

of course, only part of the question involved in the whole protection problem. In most instances the scattered radiation which must be excluded from personnel covers a large area of the barrier wall and scattering from very wide angles must be considered. A complete experimental study of this nature requires the construction of full scale walls, and walls placed at several distances from the source of radiation. This, unfortunately, could not be carried out.

It is well to point out that the recent development of compact roentgen-ray machines operating at 1,000 kv., and over, in the investigation of metal welds and castings will eventually be utilized in roentgen therapy installations. In such cases the

useful roentgen-ray beam may be pointed in almost any direction and consequently adequate protection of the operating personnel will require the construction of walls, floors, and ceiling of sufficient thickness. It remains to be seen whether it will be economically feasible to construct concrete walls of somewhat smaller thicknesses covered with lead sheeting. The latter, from a physical standpoint, might prove to be a more efficient absorber of the radiation scattered by the main concrete wall.

SUMMARY

The absorption of narrow beams of 1,000 kilovolt (peak) roentgen rays by thick layers of lead, concrete, and water has been experimentally determined. A simple experiment is described which illustrates the effect of scattering of a broad beam as it penetrates thick barriers of concrete, such as are commonly used to protect personnel.

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THREE AND ONE-HALF YEARS' EXPERIENCE WITH THE 1,000 KILOVOLT ROENTGEN THERAPY UNIT AT MEMORIAL HOSPITAL*

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FROM August 1, 1939, to February 1, 1943, 375 patients with cancer were treated by the 1,000,000 volt machine at Memorial Hospital. A study was made of the records of only those patients, 315 in number, who had finished their treatments by November 15, 1942.

Although it is too early to draw any conclusions as to the survival rate of patients treated by the 1,000,000 volt machine, nevertheless the analysis of these records revealed certain advantages and disadvantages of this unit as compared with the 200,000 volt machine.

This paper, therefore, is an account of what happened to 315 patients with cancer who were treated by a 1,000,000 volt machine, and is an expression of opinion as to the value of this higher unit in roentgen therapy.

The machine in use at Memorial Hospital consists of a low frequency resonance transformer in which is placed a coaxially mounted multi-section roentgen tube. Both the transformer and the roentgen tube are contained in a grounded steel tank. Freon 12 gas is used for insulation. The roentgen rays are generated from a target mounted in the end of an extension chamber projecting out from the bottom of the grounded tank.

The unit is simple, compact, and free from exposed high voltage. A considerable degree of flexibility has been gained through a device† which permits an adjustment of the radiation beam on a vertical plane at any angle from vertical to horizontal.[1] This flexibility is accomplished, in part, by surrounding the roentgen-ray source with an 11.2 cm. layer of mercury in a suitable steel tank in which a hollow truncated steel cone is so placed as to provide a "window" in the mercury through which the roentgen-ray beam may pass at the desired angle.

Thus the great objection which many have had to the supervoltage unit, its inflexibility, has been largely overcome, for at Memorial Hospital the machine can be adjusted to the patient, not the patient to the machine.

The importance of this careful positioning of the roentgen-ray beam with respect to the lesion to be treated cannot be overestimated. This is especially true when small beams are used, as they are in the higher voltage unit, because of the relatively larger gain in depth dose.

The chosen position of patient and roentgen-ray beam should be maintained throughout the treatments. The added flexibility of angulations of the beam enables the radiologist to make the desired setup with the patient in a comfortable position, thus lessening the possibility of the patient changing his position during the course of the treatment.

To make doubly sure that the beam is at all times properly directed, the patient is under constant observation by the technician through a window, which, while providing a full view of the treatment room, at the same time affords the technician complete protection from roentgen rays. This window‡ consists of a glass "aquar-

† Designed by Dr. G. Failla.

‡ Designed by Dr. G. Failla.

* Read in part at the Forty-second Annual Meeting, American Roentgen Ray Society, Cincinnati, Ohio, Sept. 23–26, 1941.

TABLE I

CLASSIFICATION OF PATIENTS WITH CANCER

	Total number of cases	Suitable	Unsuitable
Lung	93	59	34
Esophagus	62	39	23
Stomach	29	9	20
Bladder	25	13	12
Prostate	17	7	10
Rectum	17	14	3
Cervix and vagina	17	6	11
Ovary	13	5	8
Bone	9	8	1
Miscellaneous*	33	10	23
Total	315	170	145

* The miscellaneous group includes patients with retroperitoneal sarcoma, Hodgkin's disease, lymphosarcoma, and those with cancer of the pancreas, uterus, gallbladder, tonsil, kidney and parotid gland.

ium," filled with water, which fits closely in an opening in the concrete wall of the treatment room.

Still another device has been provided to insure accuracy of dosage. It consists of an integrating and recording dosimeter.* It provides a graphic record of each treatment. The ionization chamber of this instrument is only 0.63 cm. in diameter and 1.5 cm. in length. Because it is so small it records the dose delivered to the skin, including back scatter. The chamber is connected to the measuring device by means of two flexible cables about 0.5 cm. in diameter and 15 feet in length. It is readily attached to the patient at the center of the field to be irradiated. If the patient were to move during the treatment, the chamber would be displaced with respect to the roentgen-ray beam, and if the movement were considerable, it would show on the graphic record.

By these several devices, the flexibility of the machine and the accuracy of the beam are made possible.

At the Memorial Hospital the following factors are used with the 1,000,000 volt machine: a tube current of 3 ma.; a filter consisting of 2 mm. tungsten, 2 mm. mercury, 8 mm. copper, and several millimeters of water. These form a half-layer value of 3.8 mm. of lead. The target skin distance is 70 cm. The intensity of the vertical beam is 50 r per minute.

No selection of patients was made for treatment by the 1,000,000 volt machine. The majority, however, were in the advanced stages of the disease, since those patients who have failed to improve under other treatments are often the first to be subjected to new methods of treatment.

Of these 315 patients, 145 were unsuitable for use as a means of judging the value of the treatment. They were either too sick to finish even the original outlined course of treatments, or had proved liver or peritoneal metastases, or on first examination revealed widespread metastases to bones or lymph nodes. The remaining 170 patients, many with inoperable or recurrent cancer, were classified as suitable.

The data here presented, however, include all patients, suitable and unsuitable.

In Table I, the 315 patients are classified according to the form of the disease.

Ninety-nine of the 315 patients had received previous treatment, either surgical or radiation.

In only 32 patients of the 315 was there no microscopic proof of disease. Of these 32 patients, 10 had recurrent disease which had been proved formerly by biopsy; and 4 had gross evidence of inoperable cancer, revealed by exploratory operation. No biopsy was taken of these 4, however, as it was considered unnecessary to do so. In only 18 of the entire series of 315 patients, therefore, was the disease histologically unproved.

The treatments were given in daily fractional doses of 250 to 400 r, measured in air. As a rule the patients tolerated them better than similar patients under the 200,000 volt machine. Roentgen sickness was less; it was not even a problem. With a few exceptions the skin reaction from a total dose ranging from 2,100 to 5,000 r per portal was not severe.

* Designed by Dr. G. Failla and the Leeds and Northrup Company.

The actual tumor dose in roentgens was calculated by the usual method. The depth dose data were taken from measurements made by Quimby and Focht[6] on the machine used in these treatments.

An analysis of the groups presented in Table I follows.

The largest groups were made up of those treated for cancer of the lung and of the esophagus. In fact, during this three and one-half year period all patients, with few exceptions, who were admitted to the hospital with one or the other of these diseases were treated by the 1 million volt unit.

CANCER OF THE LUNG

Of the 93 patients treated for cancer of the lung, 21 are alive, 68 are dead, and 4 have been lost track of. The survival rate is shown in Table II.

The cases of lung cancer were usually treated through two circular fields, one anterior and one posterior. The size of the fields varied with the size of the tumor. At times a lateral field was added. The daily dose ranged from 250 to 400 r and the total dose for each field from 4,000 to 5,200 r.

The following case report shows the favorable result from the use of the 1 million volt unit in treatment of cancer of the lung.

Case 1. *Diagnosis:* Superior sulcus bronchogenic carcinoma with rib destruction.

Histopathological Diagnosis: Bronchogenic cancer.

Treatment: The patient was treated through an anterior and a posterior field each receiving 300 r to one field daily with a total dose of 4,500 r to each circular field 11.5 cm. in diameter The tumor dose was 4,500 r (Fig. 1).

Subsequent Course: The condition of the patient is excellent. The roentgenograms of the chest, fifteen months after finishing the treatment, show complete regression of the tumor (Fig. 2, *A* and *B*).

CANCER OF THE ESOPHAGUS

Of the 62 patients treated for cancer of the esophagus, 11 are alive, 47 are dead, and 4 have been lost track of.

The survival rate is shown in Table III.

The esophageal cancers were treated through four fields, two anterior and two posterior. The size of the field was usually 14 by 7 cm. The patient received from 300 to 400 r to each field daily, with an average total dose ranging from 3,600 to 4,200 r for each field.

The following case illustrates the favorable result of this treatment.

Table II

CANCER OF THE LUNG

Alive: 21				
More than 2 yr.	Two yr.	More than 1 yr.	Between 6 and 12 mo.	Between 3 and 6 mo.
4 (2 N.E.D.*) (2 with disease)	1 (with disease)	6 (5 N.E.D.) (1 with brain metastases) but primary controlled	6 (3 N.E.D.) (2 with disease) (1)?	4 (1 N.E.D.) (3 too soon after finishing treatment to be proved)
Dead: 68				
After a period of more than 2 yr.	After a period of 2 yr.	After a period of more than 1 yr.	After a period between 3 and 12 mo.	After a period between 3 days and 3 mo.
0	0	9	32	27

* N.E.D. = No evidence of disease.

CASE II. *Diagnosis:* Cancer of esophagus.

Histopathological Diagnosis: Epidermoid carcinoma, Grade 3.

Treatment: The patient was treated through four fields, two anterior and two posterior. The size of the field was 14 by 7 cm. He received a total dose of 4,200 r to each field with a tumor dose of 8,000 r (Fig. 3).

Subsequent Course: The patient who had been able to swallow only liquids is now able to eat everything without difficulty. He is in excellent general condition. The roentgenograms, nine months after treatment, show a normal esophagus (Fig. 4, *A*, *B*, *C*, *D*).

The result of the esophagoscopic examinations before and after treatment is as follows:

May, 1942, before treatment. The esophagoscope was passed with difficulty and at a distance of 18 cm. from the incisor teeth there was a fungating ulcerated lesion which was most prominent on the posterior lateral walls. It seemed to obstruct the esophagus almost completely.

September, 1942, after treatment. The esophagoscope was passed without difficulty and at a distance of 18 cm. from the incisor teeth a stricture was noted. There was no bulging within the lumen. Although the mucosa

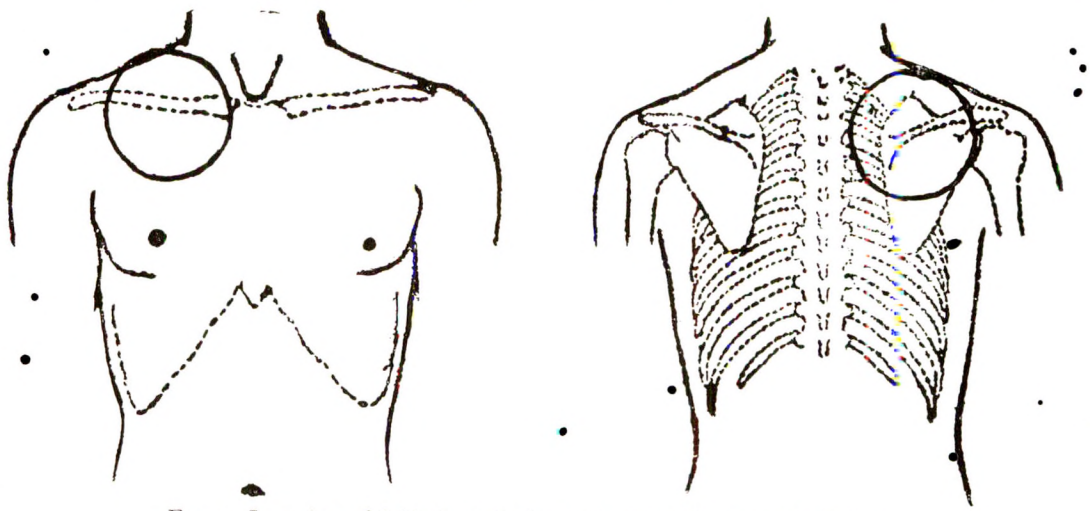


FIG. 1. Location of fields in radiation treatment of cancer of the lung

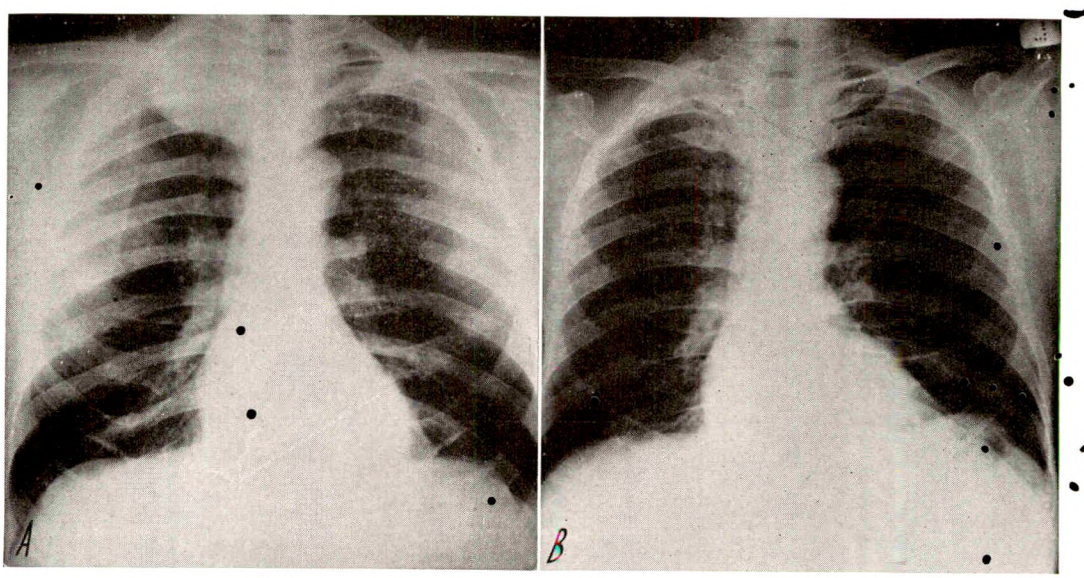


FIG. 2. Case 1. Cancer of the lung. *A*, roentgenogram of the chest taken before radiation treatment. *B*, roentgenogram of the chest taken fifteen months after completing treatment.

TABLE III

CANCER OF THE ESOPHAGUS

Alive: 11			
More than 2 yr.	More than 1 yr.	Between 12 and 6 mo.	Between 6 and 2½ mo.
0	2 (N.E.D.)	4 (N.E.D.)	5 (2 with disease) (1 N.E.D.) (2 too soon after finishing treatment to be proved)

Dead: 47			
After a period of more than 2 yr.	After a period of more than 1 yr.	Between 12 and 6 mo.	Between 3 mo. and 1 day
1	2	17	27

was somewhat inflamed, no ulceration could be seen. There was an excellent regression of the tumor from the therapy. The lumen appeared adequate.

During the course of treatment, both the lung and esophagus cancers were observed by endoscopic methods.

GASTRIC CANCER

Of the 28 patients treated for gastric cancer, 2 are alive, 21 are dead, and 5 have been lost of track of.

The survival rate is shown in Table IV.

No patient in this group was classified as "primary operable," and then irradiated instead of receiving surgery.

The 2 living patients have recurrent disease.

Of the 21 patients who died, 19 had an exploratory laparotomy done which showed that 7 had proved metastases to the liver, pancreas, peritoneum or omentum; that 9 must be considered inoperable without giving detailed reasons; and that 3, with recurrent disease, were inoperable. This leaves only 2 that were called inoperable by roentgen and clinical studies alone.

It is evident, therefore, that the results from this group furnish little evidence of the possibilities of million volt therapy in the treatment of gastric cancer.

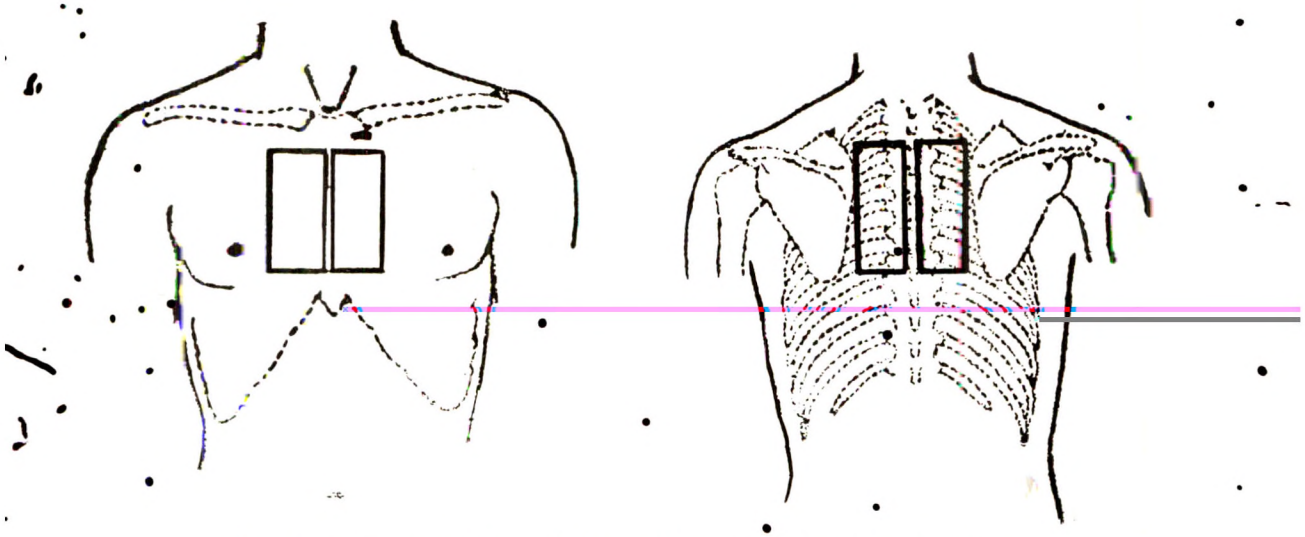


FIG. 3. Location of fields in radiation treatment of cancer of the esophagus.

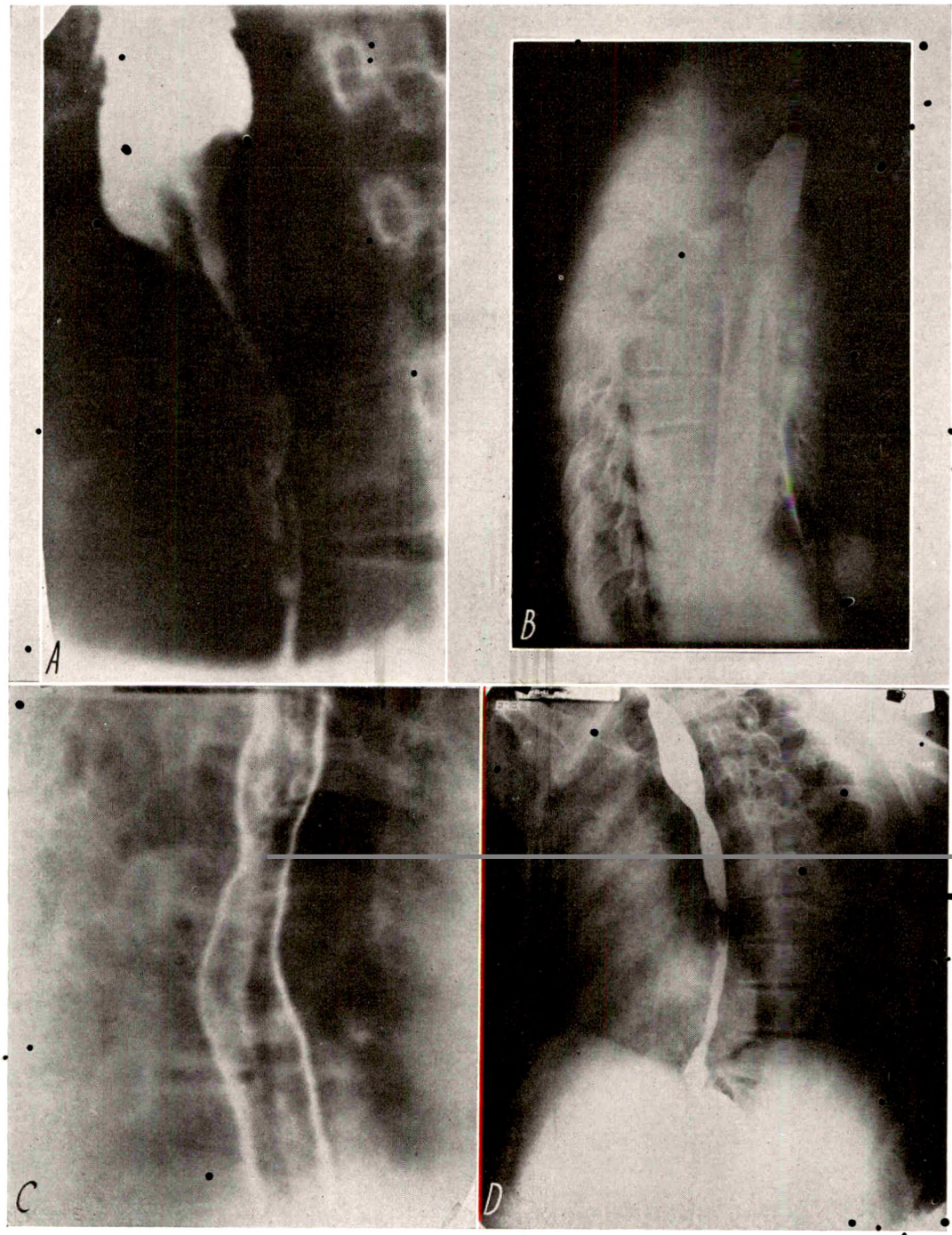


FIG. 4. Case II. Cancer of the esophagus. *A* and *B*, roentgenograms of the esophagus taken before radiation treatment. *C* and *D*, roentgenograms of the esophagus taken nine months after completing treatment.

CANCER OF THE BLADDER

Of the 25 patients treated for cancer of the bladder, 7 are alive, 16 are dead, and 2 have been lost track of.

The survival rate is shown in Table V.

It has been our experience in the radiation therapy of bladder cancer with either 200,000 or 1,000,000 volts, that if the tumor

TABLE IV

GASTRIC CANCER

Alive: 2				
More than 2 yr.	2 yr.	More than 1 yr.	Between 12 and 3 mo.	Between 3 and 1 mo.
—	—	—	2	—
Dead: 21				
After a period of more than 2 yr.	2 yr.	More than 1 yr.	Between 12 and 6 mo.	Between 6 mo. and 2 wk.
—	—	—	4	17

is infected and the patient has urgency and frequency of urination, roentgen therapy affects the patient in one of two ways: either the symptoms subside rapidly during the first treatments, indicating a favorable response; or the symptoms increase, often to the point where irradiation has to be discontinued. If the patient is affected seriously by the treatment, the symptoms appear to be increased earlier and with greater severity when the higher voltage is used. This may be due to the greater depth dose.

CASE III. *Diagnosis:* Cancer of the bladder.

Histopathological Diagnosis: Papillary carcinoma.

The cystoscopic report before the treatment was as follows: The cystoscope reveals a large papillary, fungating lesion which involves most of the neck of the bladder. It appears to be an extremely extensive neoplasm.

Treatment: The patient was treated through five fields, one anterior, two lateral and two posterior, each field being 14 by 11.5 cm. The daily dose was 300 r to each field. The total dose was 3,000 r to the anterior and posterior fields and 3,600 r to the lateral fields. The tumor dose was 6,060 r.

Subsequent Course: The patient is in excellent health with no bladder symptoms. The cystoscopy one year and nine months after the treatments were finished showed only "scarring" in the vault. There was no evidence of recurrence.

CANCER OF THE PROSTATE

Patients with cancer of the prostate are often treated at Memorial Hospital with stilbestrol, omitting irradiation. This ac-

TABLE V

CANCER OF THE BLADDER

Alive: 7				
Three yr.	More than 2 yr.	More than 1 yr.	Between 12 and 6 mo.	Between 6 and 3 mo.
1 (N.E.D.)	2 (N.E.D.)	1 (N.E.D.)	1 small recurrence; treated with gold radon seeds	2 (N.E.D.)
Dead: 16				
—	—	3	4	9 (1 patient died of pneumonia)

TABLE VI

CANCER OF THE PROSTATE

3 yr.	More than 2 yr.	2 yr.	More than 1 yr.	Less than 1 yr.
Alive: 7				
1 (N.E.D.)	3 (1 N.E.D.) (2 some recurrent disease) Bilateral orchidectomy recently	1 (N.E.D.) Stilbestrol since treatment	2 (1 No evidence of active disease, stilbestrol also) (1 with disease)	—
Dead: 8				
—	—	—	—	8 (1 committed suicide)

counts for the small number treated by the 1,000,000 volt unit.

Orchidectomy and stilbestrol are well known and many are enthusiastic over this new method of treating cancer of the prostate. For this reason roentgen therapy is neglected. This is a mistake, for irradiation is a valuable adjunct in the treatment, especially when bone metastases are present.

Of the 17 patients treated for cancer of the prostate, 7 are alive, 8 are dead, and 2 have been lost track of.

The survival rate is shown in Table VI.

The patients in this group were treated

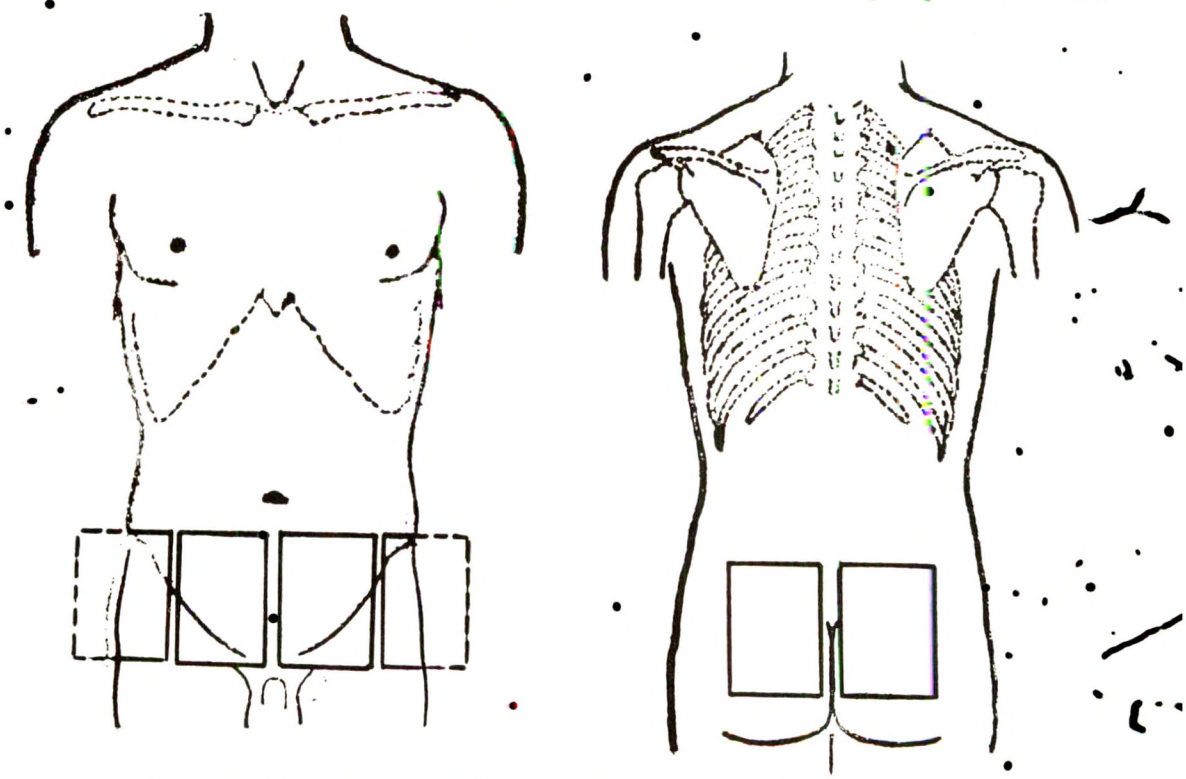


FIG. 5. Location of fields in radiation treatment of cancer of the prostate.

through six or seven fields, two anterior, two posterior, two lateral and some with a perineal field. The daily dose was usually 300 r to one field. The total dose was 3,000 r to each of the fields (Fig. 5).

CASE IV. *Diagnosis:* Cancer of the prostate.

Histopathological Diagnosis: Cancer of the prostate. The prostate was enlarged, irregular, and stony hard.

Treatment: The patient was given 3,300 r to each of the two anterior fields, 3,300 r to each of the two posterior fields and 2,100 r to each of the two lateral fields. In addition, 2,100 r was given through a perineal field. The tumor dose was 7,120 r.

Subsequent Course: The patient is in excellent condition three years after completion of treatment. He is working steadily and shows no evidence of disease. The rectal examination revealed a rather elastic prostate not at all typical of active disease.

CANCER OF THE RECTUM

Of the 17 patients treated for cancer of the rectum, 6 are alive and 11 are dead.

The survival rate is shown in Table VII.

dose was between 300 and 400 r per field, with a total dose of 3,000 to 3,600 r per field.

CASE V. *Diagnosis:* Cancer of the rectum.

Histopathological Diagnosis: Adenocarcinoma.

The rectal examination revealed a large fixed angular carcinoma beginning 1 cm. from the skin margin. The lumen was greatly narrowed.

Treatment: The patient was treated in the manner explained above, with a total tumor dose of 7,150 r.

Subsequent Course: The patient is in good general condition and today, one year and nine months after finishing the treatments, shows no evidence of disease. The lumen is sufficient for evacuation.

Not much can be reported on the other cases of this group. Although as a rule rectal cancer does not respond completely to roentgen therapy, yet here are 3 cases that show at least a very good regression of the disease. Two of these were inoperable at the time the treatments began and the other one, while operable as far as the local lesion was concerned, was inoperable because of the patient's advanced years.

TABLE VII

CANCER OF THE RECTUM

Alive: 6				
More than 2 yr.	2 yr.	More than 1 yr.	1 yr.	Between 10 and 12 mo.
1 N.E.D.	—	3 (1 No evidence of active disease) (2 with disease)	—	2 with disease

Dead: 11				
More than 2 yr.	2 yr.	More than 1 yr.	1 yr.	Between 12 and 3 mo.
—	—	3 (1 had ovarian cancer and died of this)	—	8

The arrangement of the fields in patients with cancer of the rectum is similar to that in patients with cancer of the bladder or prostate. Six pelvic fields, about 14 by 11 cm. in size, were generally used. The daily

CANCER OF THE CERVIX AND VAGINA

Of the 17 patients treated for cancer of the cervix and vagina, 3 are alive, 10 are dead, and 4 have been lost track of.

The survival rate is shown in Table VIII.

TABLE VIII

CANCER OF THE CERVIX AND VAGINA

Alive: 3				
More than 2 yr.	2 yr.	More than 1 yr.	Between 12 and 4 mo.	Less than 4 mo.
—	—	—	3	—
Dead: 10				
—	1	4	5	—

It is significant that of this group only 2 patients had not been thoroughly treated previously by surgery, roentgen rays, radium or all three; and that 11 of the 17 were definitely unsuitable on first examination for judging the effectiveness of million volt therapy.

Since nearly all of the patients in this group had received previous roentgen irradiation a fixed plan of treatment could not be followed. Whenever it was possible the patient was treated through six pelvic fields. The total dose was approximately 2,600 r to each field. The previous treatments made it impossible to give a higher total dose; it was often lower.

CANCER OF THE OVARY

Of the 13 patients treated for cancer of the ovary, 3 are alive and 10 are dead.

The survival rate is shown in Table IX.

Eight of the 13 patients in this group were, on first examination, considered as unsuitable. All in the group had received previous surgery or roentgen treatments.

For the past three months all patients with ovarian cancer have been treated by the 1,000,000 volt machine. It is too early to give any report of them. The patient is treated through four pelvic and four abdominal fields with an average of 200 r to two fields daily and a total dose of 3,000 r to each field.

BONE SARCOMA

Of the 9 cases with bone sarcoma 3 are alive, 5 are dead, and 1 has been lost track of.

The survival rate is shown in Table X.

On 2 of these patients, amputation was performed after completion of irradiation.

TABLE IX

CANCER OF THE OVARY

Alive: 3				
More than 2 yr.	2 yr.	More than 1 yr.	Between 12 and 8 mo.	Less than 8 mo.
—	—	1 with disease	2 (1 N.E.D.) (1 with disease)	—
Dead: 10				
More than 2 yr.	2 yr.	More than 1 yr.	Between 12 and 8 mo.	Between 8 and 2 mo.
—	2	1	2	5

TABLE X

BONE SARCOMA

Alive: 3				
More than 2 yr.	2 yr.	More than 1 yr.	Between 12 and 6 mo.	Less than 6 mo.
1 (N.E.D.)	—	—	2 (No evidence of active disease)	—

Dead: 5				
More than 2 yr.	2 yr.	More than 1 yr.	Between 12 and 6 mo.	5 mo.
—	—	—	3	1

In one, a Ewing's sarcoma, there was no microscopic evidence of residual disease although the original biopsy was positive; in the other, an osteogenic sarcoma, a microscopic examination revealed marked irradiation effects.

SUMMARY AND CONCLUSION

The results of the treatments, as shown in Table XI, are at first glance discouraging. Of a total of 315 patients, 213 are dead.

However, the disease was so advanced in 145 patients that palliation was all that could be expected. If we subtract these 145 unsuitable cases from the 315, only 170 remain. Even many of this group presented more complicated problems than does the usual patient admitted for radiation therapy. Some had already been treated up to the skin tolerance by the 200,000 volt unit, some were so sensitive to irradiation that it was only after their treatments by the 200,000 volt unit had been interrupted several times that they were referred for treatment with the higher unit; and some were so sick that the course of treatments had to be stopped before a satisfactory amount of radiation was given.

TABLE XI

A SURVEY OF THE PATIENTS TREATED

Diagnosis	Number of Cases	Previous Treatment	Pathological Condition Unproved	Dead	Alive	Result Unknown
Cancer of lung	93	19	7	68	21	4
Cancer of esophagus	62	15	3	47	11	4
Cancer of stomach	29	12	5	21	2	6
Cancer of bladder	25	9	3	16	7	2
Cancer of prostate	17	7	3	8	7	2
Cancer of rectum	17	4	3	11	6	0
Cancer of cervix	14	12	1	8	3	3
Cancer of vagina	3	3	0	2	0	1
Cancer of ovary	13	13	0	10	3	0
Bone sarcoma	9	2	1	5	3	1
Miscellaneous	33	23	6	17	8	3
Total	315	119	32	213	71	31

Moreover, the million volt therapy probably resulted in palliation for many patients over a period of months and even years before they died.

Physical measurements show that at greater depth the higher voltage does deliver a larger dose than is possible with the lower unit. Moreover, when small fields are used the increased depth dose becomes even greater since there is little scattered radiation. This advantage is lessened when the cross-fire technique is used with the higher voltage unit. The dose on the surface of exit somewhat offsets the practical value of the greater depth dose.

The million volt unit, however, does permit the use of a small field. This is an apparent advantage, for in the clinical application of roentgen radiation a field should be used that will include the tumor in its entirety with as little of the surrounding normal tissue as possible. Another advantage is the relatively greater depth dose in the smaller fields in comparison to the 200,000 volt machine.

The present analysis would seem to indicate, however, that the million volt machine is not a substitute for the 200,000 volt unit; that its use is limited to the treatment of deep-seated tumors.

But at a depth of 7 or 8 cm., the lower voltage unit gives nearly as great a dose as the higher voltage unit, and since the former is a more flexible machine it is better to use it in the treatment of tumors within that range. On the other hand, in the treatment of deep-seated tumors, especially those near vital organs, the higher voltage unit is better.

It will be interesting at some later date to study the results of treatments given by the million volt machine to patients who were not in such an advanced stage of disease and thus more suitable for judging the results of roentgen therapy.

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Twenty-eighth Annual Meeting: 1944, to be announced.

EDITORIAL

ROENTGENOLOGICAL PROBLEMS, PAST AND FUTURE

RECENTLY Barclay,* in an address before the Faculty of Radiologists, in reviewing a number of the pitfalls in the history of roentgenological diagnosis, analyzed some of the lessons which have been learned over these many years and with prophetic vision pointed out a road to the future. No one is perhaps better equipped than Barclay as he has stood at the forefront of his profession from the beginning of his interest in roentgenologic work. He can therefore with some feeling point to the mistakes in roentgenologic practice which have caused setbacks and lost ground which has been recovered only by the subsequent great contributions to medicine which have been brought about through roentgen-ray studies.

In his reminiscences, Barclay recalls that from its very beginning the roentgen ray was used as a method of study of the pathologic and as a result the roentgenologist was always looking for the abnormal, failing to appreciate to the fullest extent that he was also observing the normal. His well grounded teachings in morbid anatomy gave the roentgenologist fixed ideas and when he actually saw living anatomy as demonstrated by the roentgen ray he, in his early work, assumed that these findings must be abnormal. The introduction of the opaque meal method of examination of the alimentary tract coincided with the decline in popularity of Glénard's disease or splanchnoptosis, a diagnosis which had been very much in vogue for a number of years. The roentgenologic method of study revived interest in this condition and quite naturally the roentgenologist was called into consultation and, lacking a knowledge of the variations in the normal, he quite easily fell into the unsuspecting mistake of interpreting the normal as being abnormal and undoubtedly contributed unwittingly to the tremendous vogue which splanchnoptosis had for a number of years, and wittingly or not, was a party to the surgical vogue of the times. As some one has expressed it, everything that was fixed in the abdomen the surgeon loosed and all that was loosed he fixed.

Little did the roentgenologist at the time realize there were certain normal patterns for individuals of different habitus and it was the work of Barclay that contributed so much to the elucidation of such observations. His studies of the gastrointestinal tract are epoch making in that respect and it was through the efforts of such men as Barclay that roentgenologists finally passed from the stage of having a fixed idea from the traditional teaching of anatomy as regards the position of the abdominal viscera.

Roentgenologists attempted to find out for themselves the anatomy of the normal but in order to do this the studies had to be carried out in the course of their routine work on patients who were considered to be normal, and such other normal subjects as would collaborate. Barclay points out feelingly that if anatomists had only appreciated the potential value of roentgenologic methods and at once applied them to the anatomical study of the viscera in the normal, living subject, this dark period of uncertainty regarding the normal anatomy of the gastrointestinal tract would have quickly ended.

The schools of physiology were also remiss in their full appreciation of the value of roentgenology as an aid in studying the gastrointestinal tract. The physiological teaching at that time suggested that the alimentary tract was a tube, almost a test tube, in which certain definite actions and movements took place. The idea was even propagated that everything should work according to a fixed schedule and should

* Barclay, A. E. Radiology—empiricism or science? *Brit. J. Radiol.*, November, 1943, *16*, 344–347.

this schedule by any chance not be adhered to, stasis would occur and various toxic products would be absorbed, and many of the patient's symptoms were explained on this ground. As a result of such teaching, constipation, Barclay says, became the bogey of the day and there was ushered in a period in which stasis in the gastrointestinal tract was considered the causative factor of many ills of everyday life, particularly arthritic conditions. There were certain areas that were commonly thought to be particularly prone to stasis, namely the terminal ileum, the cecum and various parts of the large intestine, and again the roentgenologist being unfamiliar with the normal, quite easily fell into this conception of stasis.

Not only was the early roentgenologist led astray in the interpretation of the gastrointestinal tract but also in the studies of the chest. The poor quality of the roentgenograms in the beginning of the roentgen methods of study possibly contributed no little to the errors which trapped the roentgenologist. In addition, few roentgenologists having had the opportunity to study the normal misinterpreted the shadows at the roots of the lung which stood out so prominently, mistaking them for enlarged lymph nodes caused from living in the dusty streets of the city, or lymph nodes of tuberculous origin, whereas in fact, as we know now, these were merely the normal vascular markings of the roots of the lungs. With each improvement in the technique there was an added burden of the interpretation of the normal lung structure and, as Barclay points out, "a shadow is not an entity in itself and has no characteristics of its own. It is merely the image of some object which lies between it and the source of the rays." Thus the recognition and interpretation of such structures depends entirely on a knowledge of the features of the structures themselves.

To the roentgenologist who is well acquainted with the normal anatomy of the lungs, the wealth of detail provided in good chest roentgenograms of today proves of great value but to the inexpert it may equally prove a stumbling block in that he may read into the normal structures pathological conditions.

With each advancement in the technical procedures of roentgenology there is an added burden placed on the roentgenologist. How may we meet this advance? It is a well known fact that practically no or very little roentgenological instruction is now included in the teaching of anatomy. While every first class hospital has an adequate department of roentgenology there is practically no school of anatomy which uses the roentgen ray in its courses of instruction, and the physiological departments in the various universities are completely lacking in roentgenological equipment for teaching the students the normal physiological movements of the gastrointestinal tract, to say nothing of the wealth of detail that might be taught concerning the physiology of the lungs by the use of the roentgen ray.

Barclay, with prophetic vision, points to the need of well equipped research departments in the universities closely allied with the departments of anatomy, physiology and clinical medicine, having access also to the department of radiology of the hospital. Such research institutes, in his opinion, should be staffed by men with clinical experience in roentgenologic work. Such men with sufficiently endowed institutes could add enormously to our knowledge of the normal and perhaps could advance medicine more rapidly by such research on the normal. The necessity for such an institute is the more important as the result of the introduction of mass miniature roentgenography and the enthusiasm with which it has been received both by the profession and by the laity. As is pointed out in Barclay's discussion, the roentgenologist in such mass survey work is interpreting the normal and the diagnosis of health is infinitely more difficult and more important than that of disease.

The need for such a research institute can scarcely be doubted. What school or university will seize the opportunity to establish itself in the vanguard of such epoch-making studies?

SOCIETY PROCEEDINGS, CORRESPONDENCE AND NEWS ITEMS

Items for this section solicited promptly after the events to which they refer.

MEETINGS OF ROENTGEN SOCIETIES*

UNITED STATES OF AMERICA

AMERICAN ROENTGEN RAY SOCIETY
Secretary, Dr. H. Dabney Kerr, University Hospital, Iowa City, Iowa. Annual meeting: To be announced.

AMERICAN COLLEGE OF RADIOLOGY
Secretary, Mac F. Cahal, 540 N. Michigan Ave., Chicago, Ill.

SECTION ON RADIOLOGY, AMERICAN MEDICAL ASSOCIATION
Secretary, Dr. J. T. Murphy, 421 Michigan St., Toledo, Ohio. Annual meeting: 1944, to be announced.

ARKANSAS RADIOLOGICAL SOCIETY
Secretary, Dr. J. S. Wilson, Mack Wilson Hospital, Monticello, Ark. Meets every three months and also at time and place of State Medical Association.

RADIOLOGICAL SOCIETY OF NORTH AMERICA
Secretary, Dr. D. S. Childs, 607 Medical Arts Bldg., Syracuse, N. Y. Annual meeting: 1944, to be announced.

RADIOLOGICAL SECTION, BALTIMORE MEDICAL SOCIETY
Secretary, Dr. Walter L. Kilby, Baltimore. Meets third Tuesday each month, September to May.

SECTION ON RADIOLOGY, CALIFORNIA MEDICAL ASSOCIATION
Secretary, Dr. J.D. Coate, 434 Thirtieth St., Oakland, Calif.

RADIOLOGICAL SECTION, CONNECTICUT MEDICAL SOCIETY
Secretary, Dr. Max Climan, 242 Trumbull St., Hartford, Conn. Meets bi-monthly on second Thursday, at place selected by Secretary. Annual meeting in May.

SECTION ON RADIOLOGY, ILLINOIS STATE MEDICAL SOCIETY
Secretary, Dr. H. W. Ackemann, 321 W. State St., Rockford, Ill.

RADIOLOGICAL SECTION, LOS ANGELES COUNTY MEDICAL ASSOCIATION
Secretary, Dr. Donald R. Laing, 65 North Madison Ave., Pasadena, Calif. Meets on second Wednesday of each month at the County Society Building.

RADIOLOGICAL SECTION, SOUTHERN MEDICAL ASSOCIATION
Secretary, Dr. Roy G. Giles, Temple, Texas.

BROOKLYN ROENTGEN RAY SOCIETY
Secretary, Dr. Leo Harrington, 880 Ocean Ave., Brooklyn, N.Y. Meets monthly on fourth Tuesday, October to April.

BUFFALO RADIOLOGICAL SOCIETY
Secretary, Dr. Joseph S. Gian-Francheschi, 610 Niagara St., Buffalo, N. Y. Meets second Monday of each month except during summer months.

CHICAGO ROENTGEN SOCIETY
Secretary, Dr. Warren W. Furey, 6844 S. Oglesby Ave., Chicago, Ill. Meets second Thursday of each month October to April inclusive at the Palmer House.

CINCINNATI RADIOLOGICAL SOCIETY
Secretary, Dr. Samuel Brown, 707 Race St., Cincinnati, Ohio. Meets third Tuesday of each month, October to May, inclusive.

CLEVELAND RADIOLOGICAL SOCIETY
Secretary, Dr. D. D. Brannan, 11311 Shaker Blvd., Cleveland 4, Ohio. Meets at 6:30 P.M. at Allerton Hotel on fourth Monday each month, October to April, inclusive.

DALLAS-FORT WORTH ROENTGEN STUDY CLUB
Secretary, Dr. X. R. Hyde, Medical Arts Bldg., Fort Worth, Texas. Meetings held in Dallas on odd months and in Fort Worth on even months, on third Monday, at 7:30 P.M.

DENVER RADIOLOGICAL CLUB
Secretary, Dr. Edward J. Meister, 366 Metropolitan Bldg., Denver, Colo. Meets third Friday of each month at Denver Athletic Club.

DETROIT ROENTGEN RAY AND RADIUM SOCIETY
Secretary, Dr. E. R. Witwer, Harper Hospital. Meets monthly on first Thursday from October to May, at Wayne County Building.

FLORIDA RADIOLOGICAL SOCIETY
Acting Secretary, Dr. Walter A. Weed, 204 Exchange Bldg., Orlando, Fla. Meetings in May and November.

GEORGIA RADIOLOGICAL SOCIETY
Secretary, Dr. James J. Clark, 473 Peachtree St., Atlanta, Ga. Meets in November and at annual meeting of Medical Association of Georgia in the spring.

RADIOLOGICAL SOCIETY OF KANSAS CITY
Secretary, Dr. Arthur B. Smith, 800 Argyle Bldg., Kansas City, Mo. Meets third Thursday of each month at a place designated by the president.

ILLINOIS RADIOLOGICAL SOCIETY
Secretary, Dr. Wm. DeHollander St. John's Hospital, Springfield, Ill. Meets three times a year.

INDIANA ROENTGEN SOCIETY
Secretary, Dr. H. C. Ochsner, Methodist Hospital, Indianapolis. Meeting held the second Sunday in May annually.

KENTUCKY RADIOLOGICAL SOCIETY
Secretary, Dr. W. C. Martin, 321 W. Broadway, Louisville. Meets annually in Louisville on first Saturday in Apr.

LONG ISLAND RADIOLOGICAL SOCIETY
Secretary, Dr. Marcus Wiener, 1430-48th St., Brooklyn, N. Y. Meets Kings County Med. Soc. Bldg. monthly on fourth Thursday, October to May, 8:30 P.M.

LOUISIANA RADIOLOGICAL SOCIETY
Secretary, Dr. J. R. Anderson, 1130 Louisiana Ave., Shreveport. Meets annually during Louisiana State Medical Society Meeting.

MICHIGAN ASSOCIATION OF ROENTGENOLOGISTS
Secretary, Dr. E. M. Shebesta, 1429 David Whitney Bldg., Detroit. Three meetings a year, Fall, Winter, Spring.

MILWAUKEE ROENTGEN RAY SOCIETY
Secretary, Dr. C. A. H. Fortier, 231 W. Wisconsin Ave., Milwaukee, Wis. Meets monthly on second Monday at University Club.

MINNESOTA RADIOLOGICAL SOCIETY
Secretary, Dr. Annette T. Stenstrom, 1218 Medical Arts Bldg., Minneapolis, Minn. One meeting a year at time of Minnesota State Medical Association.

NEBRASKA RADIOLOGICAL SOCIETY
Secretary, Dr. D. A. Dowell, Medical Arts Bldg., Omaha, Nebr. Meets third Wednesday of each month, at 6 P.M. at either Omaha or Lincoln.

NEW ENGLAND ROENTGEN RAY SOCIETY
Secretary, Dr. Hugh F. Hare, Lahey Clinic, Boston, Mass. Meets monthly on third Friday, Boston Medical Library.

RADIOLOGICAL SOCIETY OF NEW JERSEY
Secretary, Dr. H. J. Perlberg, 921 Bergen Ave., Jersey City. Meets annually at time and place of State Medical Society. Mid-year meetings at place chosen by president.

NEW YORK ROENTGEN SOCIETY
Secretary, Dr. Ramsay Spillman, 115 East 61st St., New York City. Meets monthly on third Monday, New York Academy of Medicine, at 8:00 P.M.

NORTH CAROLINA ROENTGEN RAY SOCIETY
Secretary, Dr. Major Fleming, Rocky Mount, N. C. Annual meeting at time and place of State Medical Society. Mid-year scientific meeting at place designated.

NORTH DAKOTA RADIOLOGICAL SOCIETY
Secretary, Dr. L. A. Nash, St. John's Hospital, Fargo. Meetings held by announcement.

* Secretaries of Societies not here listed are requested to send the necessary information to the Editor.

CENTRAL NEW YORK ROENTGEN RAY SOCIETY
Secretary, Dr. C. F. Potter, 820 S. Crouse Ave., Syracuse. Three meetings a year. January, May, November.

OHIO RADIOLOGICAL SOCIETY
Secretary, Dr. J. E. McCarthy, 707 Race St., Cincinnati. Meets at time and place of annual meeting of Ohio State Medical Association.

PACIFIC ROENTGEN SOCIETY
Secretary, Dr. L. H. Garland, 450 Sutter St., San Francisco, Calif. Meets annually, during meeting of California Medical Association.

PENNSYLVANIA RADIOLOGICAL SOCIETY
Secretary, Dr. L. E. Wurster, 416 Pine St., Williamsport.

PHILADELPHIA ROENTGEN RAY SOCIETY
Secretary, Dr. R. P. Barden, University Hospital, Meetings first Thursday of each month from October to May inclusive at 8:15 P.M., in Thompson Hall, College of Physicians, 19 S. 22d St.

PITTSBURGH ROENTGEN SOCIETY
Secretary, Dr. R. G. Alley, 4800 Friendship Ave. Meets second Wednesday each month, 4:30 P.M., October to June, Pittsburgh Academy of Medicine.

ROCHESTER ROENTGEN RAY SOCIETY, ROCHESTER, N. Y.
Secretary, Dr. Sidney Larson, Strong Memorial Hospital. Meets monthly on third Monday from October to May, inclusive, 8 P.M. at Strong Memorial Hospital.

ROCKY MOUNTAIN RADIOLOGICAL SOCIETY
Secretary, Dr. A. M. Popma, 220 N. First St., Boise, Idaho.

ST. LOUIS SOCIETY OF RADIOLOGISTS
Secretary, Dr. P. C. Schnoebelen, 462 N. Taylor Ave. Meets fourth Wednesday of each month, except June, July, August, and September, at a place designated by the president.

SAN FRANCISCO RADIOLOGICAL SOCIETY
Secretary, Dr. H. A. Hill, 450 Sutter St., San Francisco. Meets monthly on third Thursday at 7:45 P.M., first six months at Toland Hall, second at Lane Hall.

SHREVEPORT RADIOLOGICAL CLUB
Secretary, Dr. R. W. Cooper, Charity Hospital, Shreveport, La. Meets monthly on third Wednesday, at 7:30 P.M., September to May inclusive.

SOUTH CAROLINA X-RAY SOCIETY
Secretary, Dr. T. A. Pitts, Baptist Hospital, Columbia, S. C. Meets in Charleston on first Thursday in November, also at the time and place of South Carolina State Medical Association.

TENNESSEE RADIOLOGICAL SOCIETY
Secretary, Dr. J. M. Frère, 707 Walnut St., Chattanooga, Tenn. Meets annually at the time and place of the Tennessee State Medical Association.

TEXAS RADIOLOGICAL SOCIETY
Secretary, Dr. Herman Klapproth, Sherman, Texas.

UNIVERSITY OF MICHIGAN DEPARTMENT OF ROENTGENOLOGY STAFF MEETING
Meets each Monday evening from September to June, at 7 P.M. at University Hospital.

UNIVERSITY OF WISCONSIN RADIOLOGICAL CONFERENCE
Secretary, Dr. E. A. Pohle, 1300 University Ave., Madison, Wis. Meets every Thursday from 4:00–5:00 P.M., Room 301, Service Memorial Institute.

VIRGINIA RADIOLOGICAL SOCIETY
Secretary, Dr. E. L. Flanagan, 116 E. Franklin St., Richmond, Va. Meets annually in October.

WASHINGTON STATE RADIOLOGICAL SOCIETY
Secretary, Dr. Thomas Carlile, 1115 Terry St., Seattle. Meets fourth Monday each month, October throughMay, College Club, Seattle.

X-RAY STUDY CLUB OF SAN FRANCISCO
Secretary, Dr. J. M. Robinson, University of California Hospital. Meets monthly in evening on third Thursday.

CUBA

SOCIEDAD DE RADIOLOGÍA Y FISIOTERAPIA DE CUBA
President, Dr. J. Manuel Viamonte, Hospital Mercedes, Habana, Cuba. Meets monthly in Habana.

BRITISH EMPIRE

BRITISH INSTITUTE OF RADIOLOGY INCORPORATED WITH THE RÖNTGEN SOCIETY
Medical Members' meeting held monthly on third Friday at 2:30 P.M. and Ordinary Meeting at same time on following Saturday, October to May, 32 Welbeck St., London, W.1.

SECTION OF RADIOLOGY OF THE ROYAL SOCIETY OF MEDICINE (CONFINED TO MEDICAL MEMBERS)
Meets on the third Friday of each month at 4:45 P.M. at the Royal Society of Medicine 1, Wimpole St., London, W. 1.

FACULTY OF RADIOLOGISTS
Secretary, Dr. M. H. Jupe, 32 Welbeck St., London, W. 1 England.

SECTION OF RADIOLOGY AND MEDICAL ELECTRICITY, AUSTRALASIAN MEDICAL CONGRESS
Secretary, Dr. H. M. Cutler, 139 Macquarie St., Sydney, New South Wales.

RADIOLOGICAL SECTION OF THE VICTORIAN BRANCH OF THE BRITISH MEDICAL ASSOCIATION
Secretary, Dr. Keith Hallam, St. George's Hospital, K.E.W., Melbourne, E. 4, Victoria, Australia. Meets monthly from March to November inclusive.

CANADIAN ASSOCIATION OF RADIOLOGISTS
Secretary, Dr. A. D. Irvine, 540 Tegler Bldg., Edmonton, Alberta.

SECTION OF RADIOLOGY, CANADIAN MEDICAL ASSOCIATION
Secretary, Dr. C. M. Jones, Inglis St., Ext. Halifax, N. S.

RADIOLOGICAL SECTION, NEW ZEALAND BRITISH MEDICAL ASSOCIATION
Secretary, Dr. Colin Anderson, Invercargill, New Zealand. Meets annually.

SOUTH AMERICA

SOCIEDAD ARGENTINA DE RADIOLOGIA
Secretary, Dr. Guido Gotta, Buenos Aires, Argentina. Meetings are held monthly.

CONTINENTAL EUROPE

SOCIEDAD ESPANOLA DE RADIOLOGIA Y ELECTROLOGIA
Secretary, Dr. J. Martin-Crespo, Fuencarral, 7. Madrid, Spain. Meets monthly in Madrid.

SOCIÉTÉ SUISSE DE RADIOLOGIE (SCHWEIZERISCHE RÖNTGEN-GESELLSCHAFT)
Secretary for French language, Dr. A. Grosjean La Chaux de Fonds.
Secretary for German language, Dr. Scheurer, Molzgasse Biel. Meets annually in different cities.

SOCIETATEA ROMANA DE RADIOLOGIE SI ELECTROLOGIE
Secretary, Dr. Oscar Meller, Str. Banul Mărăcine, 30, S. I., Bucuresti, Roumania. Meets second Monday in every month with the exception of July and August.

ALL-RUSSIAN ROENTGEN RAY ASSOCIATION, LENINGRAD: USSR in the State Institute of Roentgenology and Radiology, 6 Roentgen St.
Secretaries, Drs. S. A. Reinberg and S. G. Simonson. Meets annually.

LENINGRAD ROENTGEN RAY SOCIETY
Secretaries, Drs. S. G. Simonson and G. A. Gusterin. Meets monthly, first Monday at 8 o'clock, State Institute of Roentgenology and Radiology, Leningrad.

MOSCOW ROENTGEN RAY SOCIETY
Secretaries, Drs. L. L. Holst, A. W. Ssamygin and S. T. Konobejevsky. Meets monthly, first Monday, 8 P.M.

SCANDINAVIAN ROENTGEN SOCIETIES
The Scandinavian roentgen societies have formed a joint association called the Northern Association for Medical Radiology, meeting every second year in the different countries belonging to the Association.

DEPARTMENT OF TECHNIQUE

Department Editor: ROBERT B. TAFT, M.D., B.S., M.A., 103 Rutledge Ave.
Charleston, S. C.

A SIMPLE TECHNIQUE FOR CARDIAC MENSURATION*

By S. T. HERSTONE, M.D.
Instructor in Radiology and Anatomy, New York University College of Medicine
and
D. E. ZION, M.D.
Resident in Roentgen Diagnosis, Montefiore Hospital
NEW YORK, NEW YORK

ORTHODIAGRAPHY has been recognized for many years as the most accurate method of determining cardiac measurements. Despite this fact, it has not been used extensively because of several important disadvantages.

1. To trace the heart contour accurately by means of orthodiagraphy takes a period of ten to twenty minutes. In doing large series of cases this becomes extremely tedious and time consuming.

2. The patient's exposure to the roentgen ray is considerable and if oblique tracings are attempted the radiation dosage may become prohibitive.

The purpose of this paper is to describe a simple method of obtaining an orthodiagram with the elimination of these objections.

METHOD

The necessary equipment consists of a standard roentgenoscope, film, and a 10 by 12 inch cassette. The horizontal shutter of the roentgenoscope diaphragm is opened to the fullest extent. The vertical shutter is narrowed to a slit 1 cm. or less in diameter. The patient is placed with his back to the panel of the roentgenoscope so that the midline of the panel passes through the approximate center of his heart. The film is then placed immediately in front of the patient. It can be held by the patient, strapped to his chest or placed in a stand constructed for this purpose. The patient is told not to move and to breathe normally. The milliamperage is set at 4, and the kilovoltage at between 75 and 85, depending on the thickness of the chest wall. The up-and-down movement of the tube is fixed. The tube is then slowly and evenly moved from side to side, so that the narrow vertical beam emanating from it sweeps back and forth across the entire width of the chest. This movement is continued throughout the entire exposure which lasts approximately thirty seconds. In this way, the entire field is traversed about fifteen to twenty times. The film is then developed and a roentgenogram of the cardiac contour is obtained in which transverse measurements are as accurate as in the average orthodiagram.

The point of opposite pulsation on the left heart border can be identified by the use of a lead marker attached to the chest wall or to the center of the tube. If the latter method is used, a horizontal line will be seen passing through this point (Fig. 1).

DISCUSSION

This procedure has certain advantages:

1. It is an accurate means for determining cardiac measurements. For transverse measurements of the heart, it compares favorably with orthodiagraphy, and teleroentgenography. It utilizes the principle

* From the Department of Radiology, New York University, College of Medicine and the Department of Radiology, Montefiore Hospital, New York.

of orthodiagraphy and at the same time eliminates the subjective factor. It is more accurate than the 1/20 second teleroentgenogram, since it is taken through all phases of respiration and all phases of the cardiac cycle. There is less enlargement because of the use of the central beam principle.

2. The method is simple and is not time consuming. With little experience, one can complete the procedure within a few minutes, thereby diminishing the amount of exposure both to the patient and to the roentgenologist.

3. As an adjunct to roentgenoscopy it is a simple and economical method for obtaining a permanent record. It requires only a single 10 by 12 inch cassette, a film and the developing material. It can be done with equipment which is considerably less expensive than that needed for taking teleroentgenograms.

The method has certain disadvantages:

1. The vertical measurements are distorted. However, if one is interested in obtaining a true vertical measurement a procedure similar to the above may be employed, but with the horizontal shutter narrowed to a slit and the scanning performed in the vertical plane.

2. This method gives us no information as to pathological conditions in the lungs nor abnormality of cardiac movement, and because of this it should only be used as an adjunct to roentgenoscopy.

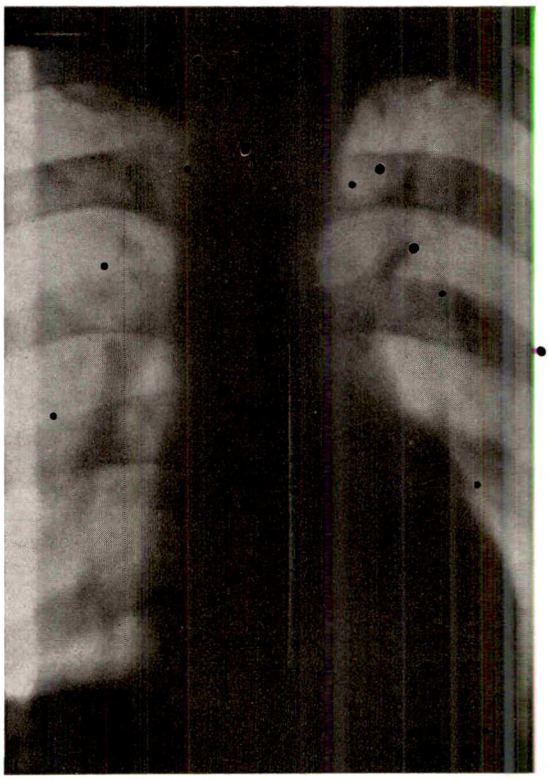


FIG. 1. Roentgenogram taken by described technique.

SUMMARY

A simple method for obtaining a permanent record of the transverse cardiac measurements is described. This method makes use of the roentgenoscope, and compares favorably with the orthodiagram and the teleroentgenogram.

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ABSTRACTS OF ROENTGEN AND RADIUM LITERATURE

ROENTGEN DIAGNOSIS

HEAD

SINGLETON, A. O. Tumors of the salivary glands, benign and locally malignant. *Surg., Gynec. & Obst.*, Feb., 1942, *74*, 569–572.

Salivary gland tumors are probably the most debatable of all tumors. They include:

(1) *Parotid Cysts.* These cysts may be congenital but they are usually secondary to chronic infection or duct obstruction. They may be symptomless but are usually marked by swelling of the gland with pain which is relieved when the ducts are opened. Treatment with roentgen irradiation is to be preferred to attempts to dissect out the cyst. If sufficient radiation is given, the gland atrophies and the cysts give no further trouble.

(2) *Mikulicz's Disease.* The name of Mikulicz's syndrome has been given to an enlargement of the salivary glands (particularly the parotid), and lacrimal glands associated with leukemia, tuberculosis, lymphosarcoma, and toxic conditions However, the condition described by Mikulicz in 1888 was not accompanied by constitutional symptoms. There is an extensive deposit of small round cells which seem to compress or displace the glandular parenchyma. The condition is very rare, not serious, and treatment is unnecessary

(3) *Adenolymphoma* (*onkocytoma*). This is one of the rarest of tumors. It is composed of a lymphadenoid stroma with germinal centers with an epithelial structure in the form of tubular glands, cysts, and papillae. It occurs in older persons, is benign, encapsulated, and grows slowly. It is not usually intimately connected with the gland and may be easily removed.

(4) *Adenoma of the Salivary Gland.* This is one of the controversial tumors. Warren states, "Adenomas readily recognized as such are extremely rare in the salivary gland." If they do occur, it is in the parotid gland. They are always encapsulated and may be cystic or solid. The differential diagnosis between the adenoma and mixed tumor is not possible before operation.

(5) *Mixed Tumors.* These tumors are benign in that they rarely metastasize but malignant in that local recurrence is a predominating characteristic. The long interest in these tumors has been from the fact that the embryology and pathology are without a satisfactory explanation and also because the treatment of them is still unsolved in spite of persistent effort on the part of the pathologist, the surgeon, and recently the roentgenologist. McFarland has estimated that 93.6 per cent of these tumors occur in the parotid gland. Transformation into carcinoma or sarcoma is extremely rare. McFarland argues against the removal of mixed tumors when they are small, claiming that recurrences are more likely in the small tumors. This, however, has not been found to be true by the author and several other investigators.

(6) *Carcinoma of the Parotid Gland.* This is one of the most incurable of the malignant tumors. Even with the most radical surgery early in the course of its growth, only a very few cures are on record.—*Mary Frances Kastine.*

LAMPE, I. Pseudo-adenomatous basal-cell carcinoma of the tongue (salivary gland tumor). *Radiology*, July, 1942, *39*, 54–61.

A case of pseudo-adenomatous basal-cell carcinoma that originated in a mixed salivary gland tumor at the base of the tongue is described. This is a relatively rare form of tumor of the tongue. The patient was a man fifty-seven years of age who was under observation and treatment for thirteen years beginning June 2, 1927. When last heard from he said he was feeling well though it had been four years since the development of pulmonary metastases. During the observation period cervical, pulmonary and bone metastases developed. Roentgen treatment reduced the size of the primary lesion and the cervical metastases disappeared. It also abolished the pain caused by the bone metastases. The lung metastases grew very slowly and caused no symptoms.

These malignant salivary gland tumors grow slowly and metastasize late. This case did not respond to initial radium treatments, doubtless because the dose was insufficient but the size of the primary lesion decreased markedly in re-

sponse to fractional roentgen therapy with a dose of moderate intensity (2,600 r, measured in air, to each of two fields over a period of a month).—*Audrey G. Morgan.*

NECK AND CHEST

MACDONALD, IAN. Mammary carcinoma. *Surg., Gynec. & Obst.*, Jan., 1942, *74*, 75–82.

The records of 2,636 cases of mammary carcinoma (1,511 five year cures and 1,125 recurrent cases) have been reviewed by the author:

Age. The age of patients with mammary carcinoma does not have the prognostic significance with which it is commonly accredited. Excluding the cases of "inflammatory" carcinoma and those occurring in gravid and puerperal women, carcinoma of the breast at any early age merits as radical an approach with as much chance of cure as in older women. The best results are obtained in those patients who are thirty-five to fifty years of age and the least favorable results are seen in the decade from fifty to sixty.

Heredity. There is evidence of an inherited susceptibility to cancer in approximately 20 per cent of this series. Among the relatives of those women having a hereditary background of cancer, there is an excess of breast cancer at least three times greater than in the general population.

Fertility. Nulliparas are more prone to develop cancer of the breast than women who have had children, but once developed, the prognosis is as good, or perhaps better, for the nulliparous woman. The number of children borne by parous women has little or no influence on prognosis.

Lactation. The most consistent apparent factor in the genesis of mammary cancer is the failure of the breast to perform its expected physiological function. A full period of lactation in childbearing women seems to offer a degree of protection against subsequent development of breast cancer.

Treatment. Early treatment is of utmost importance in the cure of certain early lesions predestined otherwise to undergo rapid growth and early dissemination. In general, however, end-results are determined more by natural selection than by early treatment. Tumors of long duration in themselves are no contraindication to radical treatment, for 25 per cent of the five year cures here recorded were treated more than one year after recognition of the tumor.

To deny patients with otherwise operable neoplasms the chance of radical treatment because of the presence of axillary node metastases is to face the possible loss of 40 per cent of attainable five year cures.

The five year post-treatment period as an arbitrary standard for the evaluation of therapeutic measures is inadequate for mammary carcinoma, and may well be replaced by a minimum nine year period.—*Mary Frances Vastine.*

BRILL, I. C., and KRYGIER, JOHN J. Primary pulmonary vascular sclerosis. *Arch. Int. Med.*, Sept., 1941, *68*, 560–577.

Two requirements are suggested for a diagnosis of primary pulmonary vascular sclerosis: (1) evidence of right ventricular hypertrophy (without any evidence of hypertrophy of the left ventricle); (2) absence of any factor commonly recognized as productive of secondary pulmonary vascular sclerosis, pulmonary hypertension or isolated strain of the right heart. The author speaks of only about 20 cases fulfilling these requirements and described in the literature. He reports 1 of these cases.

The patient was a married woman, aged twenty-one, who had been a "blue baby" and who had been considered to have "heart trouble," on several occasions. With slight exertion, she suffered dyspnea, cyanosis, precordial pain, dizziness and fainting spells. Roentgenography revealed a prominence in the region of the pulmonary artery and conus and, for that reason, a congenital heart disorder was seriously considered but there were no murmurs. Her blood pressure was 85/70; basal metabolic rate, minus 14 per cent; red blood cell count, 5,700,000 with a hemoglobin of 122 per cent and a leukocyte count of 17,550. The electrocardiogram revealed a right ventricular preponderance. The patient experienced progressive dyspnea, palpitation, precordial pain, headache, dizziness, vertigo, fainting spells and now and then vomiting. Finally, edema of the ankles developed. Shortly thereafter the patient died suddenly.

The liver and spleen were slightly enlarged and there was about 200 cc. of serosanguineous fluid in the abdomen; none in the pleural cavity. There was no lung parenchymal consolidation. Arteriosclerotic plaques were found in the pulmonary arteries. The weight of the heart was 690 grams, its left chambers being distinctly small in contrast to dilated and hypertrophied

musculature of the right ventricle and right auricle. The wall thickness of the right ventricle was 7.5 mm., while that of the left ventricle was 17 mm. The circumference of the pulmonary valve was 8.5 cm., while that of the aortic valve was 6 cm. The foramen ovale was anatomically patent but functionally closed and no congenital cardiac defects were found. The coronary vessels were free of sclerosis. Microscopically, the most striking changes included intimal proliferation of the arterioles with complete occlusion of the lumina of many of them; numerous thrombi in the arterioles—some showing recanalization, and arteriosclerotic plaques in the walls of the larger ones.

In analyzing the 20 reported cases of this condition (abstracts of the other 19 being included), the following considerations are cited: Ages varied from ten to seventy-four, 14 being under the age of forty; the most important evidence was roentgenographic—prominence of the pulmonary artery and conus, along with electrocardiographic demonstration of right axis deviation; dyspnea, orthopnea, cyanosis, fainting spells, pain in the chest, vomiting, dizziness, hemoptysis, cough, being the predominant symptoms. Most showed a low systolic pressure with a contrastingly high diastolic pressure and an elevated venous pressure. Death occurred suddenly in 5 of the 20 cases. In only 2 cases was the diagnosis made prior to death.—*A. A. de Lorimier.*

Israel, Harold L., and Hetherington, H. W. Accuracy of fluoroscopy in the detection of pulmonary tuberculosis. *Am. J. M. Sc.*, Feb., 1941, *201*, 224–232.

Since 1933, fluoroscopic examinations of the chest have been routine for the study of new admissions at the Henry Phipps Institute. Likewise, periodic examinations and screenings have thus been accomplished.

This analysis concerns 1,021 duplicate examinations including fluoroscopy and roentgenographic studies. These examinations were accomplished by three groups of physicians: one group having extensive experience in fluoroscopy and roentgenologic interpretations; one group having considerable experience in fluoroscopy but little or no training in roentgenologic interpretation, and the third group having had no more than short periods of training in either. Abnormalities were classified as: "definite," "probable," or "possible."

In only 4 out of 48 cases having easily recognizable pathologic conditions were errors made. Satisfactory results were obtained even among obese patients. Though many minimal infiltrates were missed by the inexperienced, most of these were recognized fluoroscopically by those who were well experienced. Fluoroscopy was found to be especially trustworthy in the examination of children.—*A. A. de Lorimier.*

Farinas, Pedro L. Mucosography of the respiratory tract. *Radiology*, July, 1942, *39*, 84–87.

The internal surface of the organs of the respiratory tract can be studied by coating the mucosa with a very thin layer of contrast substance. The opaque medium is vaporized at the vestibule of the larynx and inhaled in fine particles. Films taken immediately after the inhalation show the mucosa as a thin dense line which contrasts sharply with the air in the organs.

The pharynx and larynx are anesthetized with 2 cc. of a 2 per cent solution of pantocaine and the trachea with 1 cc. of a 1 per cent solution of the same anesthetic. The contrast medium—Merck's 40 per cent solution of iodipin—is vaporized with an atomizer, the curved end of which is introduced behind the epiglottis. For examination of the larynx and upper part of the trachea only 5 cc. of iodipin is required. If the lower part of the trachea and the bronchi are to be examined 15 cc. is necessary. If there are any tumors of these organs they are outlined by the contrast medium and can be seen very clearly. Illustrative roentgenograms of the trachea, larynx and bronchi are given. The method is harmless, well tolerated, does not cause any injury of the glottis and its use is simple and easy.—*Audrey G. Morgan.*

LaDue, John S. Bronchiolitis fibrosa obliterans. *Arch. Int. Med.*, Oct., 1941, *68*, 663–673.

Bronchiolitis fibrosa obliterans appears to be a rare condition caused by local injury in the walls of the bronchi. Summarizing etiological descriptions of cases reported in the literature, there would seem to be three categories: Those cases caused by inhalation of irritants such as nitric or sulphuric acid fumes, poisonous gases and the vapors of certain solvents—or such as produced because of inhalation of foreign

bodies; pathological complications by certain acute infectious diseases such as measles, influenza, scarlet fever or even chronic bronchitis as found with asthma; those cases wherein no definite etiological factors can be recognized—the so-called idiopathic group.

Microscopically, the condition is characterized by numerous miliary, possibly multangular densities with fine white streaks radiating therefrom. These nodulations may be found to represent partly destroyed bronchioles with an overgrowth of granulation tissue. Frequently, the lumina are filled with detritis consisting of desquamated epithelium, polymorphonuclear leukocytes, fibrin and blood. Later, more and more fibrous tissue replacement occurs, possibly with bronchiectatic cavitations. There may be considerable emphysema, usually of the disseminated interstitial type. These changes may produce very severe respiratory distress: dyspnea, even cyanosis; possibly fever and cough with expectoration of mucoid or even bloody sputum.

The author reports such a case—the only one discovered among 42,038 autopsies performed by members of the Department of Pathology of the University of Minnesota Medical School since 1899. The patient was a twenty year old Negro, who was brought into the hospital because of unconsciousness, dyspnea and cyanosis. He later regained consciousness and described progressive weakness, fatigue and headaches, with chronic cough and hemoptysis, all developing over a period of several months. He had a temperature of 101.2°F. His respiration rate was 42 per minute. His red cell count was only 1,990,000; hemoglobin, 38 per cent. His white cell count varied from 8,000 to 16,000 with a polymorphonuclear differential as high as 79 per cent. He was placed in an oxygen tent but died nine days later.

Clinically, his case had been considered to be one of miliary tuberculosis. Little attention had been given to the patient's occupation—and his own remarks that his trouble was due to breathing a dye (trichlorethylene). *A. A. de Lorimier.*

CHAPMAN, JOHN, and WIGGINS, JOHN A. Circumscribed and isolated bronchiectasis. *Ann. Int. Med.*, May, 1941, *14*, 2047–2057.

Bronchiectasis is generally considered to be a widespread and diffuse disease involving a greater part of a lobe or several lobes.

Among more than 400 bronchographic studies at the State Tuberculosis Sanatorium, the authors have distinguished 6 as showing bronchial dilatations limited to one or a few bronchi. Reports of only 2 such cases were found in the literature.

All of these cases were featured by relatively mild symptoms but these, persistent, with expectoration and some purulent exudate but little or no hemoptysis. In all the cases, the dilatations were saccular in type and on the right side.—*A. A. de Lorimier.*

PAGEL, WALTER, and SIMMONDS, F. A. H. Cavity healing and bronchial occlusion. *Am. J. M. Sc.*, Feb., 1942, *203*, 177–187.

There are three methods of healing of cavities in tuberculous lungs: (1) by conversion into a solid caseous focus which may eventually calcify; (2) by formation of a radiating scar obliterating the cavity, and (3) by healing, leaving an open cavity. The first two methods are alike in the final result—closure of the cavity—but they differ in pathogenesis, and one is not the terminal stage of the other; that is, solid caseous material that tends to calcification is not likely to disappear and leave a fibrotic scar.

Work done by the authors in the Pathology Department of the Central Middlesex County Hospital, London, shows that the first is by far the most common method of healing. They describe 6 cases of their own in all of which healing took place by conversion of the cavity into a solid caseous nodule. A comparison of roentgen findings before death and necropsy findings shows that four to five weeks is the minimum time required for the process. A study of these 6 cases and 5 more of their own and 33 cases from the literature confirms their theory that this is the most frequent form of healing and that calcified nodules frequently mark the terminal stage of healing of former cavities.

There is anatomical evidence that occlusion of the draining bronchi, either by obstructive caseous bronchitis or the formation of a caseous plug is an important factor in causing this form of healing.

Roentgenograms and photographs of anatomical specimens illustrating the cases are given.—*Audrey G. Morgan.*

HAUSER, HARRY. Cancer of the lung in infancy. *Radiology*, July, 1942, *39*, 33–38.

Primary cancer of the lung in infancy is extremely rare. Only 3 cases have been reported

in the American literature. The case described in this report is the only one found in an infant in more than 14,000 autopsies performed at the Cleveland City Hospital in the past twenty-eight years.

The case was in a colored infant seventeen months of age who had developed dry cough five months before. She was admitted to the hospital on account of cough and dyspnea. Roentgen examination showed homogeneous increased density of the whole left half of the lung. The heart and trachea were considerably displaced to the right. She had suppurative otitis media and acute pharyngitis, and the lung condition was diagnosed as left empyema. But on aspiration of the left pleural cavity only a small amount of fluid was obtained with some fragments of tissue which suggested malignant tumor. The child grew worse progressively and died less than a month after admission. Microscopic examination showed that the tumor was a small cell carcinoma of the left lung. Roentgenograms and photomicrographs of the findings are given.

These tumors are rare even in childhood and early adulthood. The most common error in differential diagnosis is that of massive pleural effusion. If there are large dense homogeneous shadows on one side of the chest the possibility of primary bronchogenic carcinoma should always be considered. These tumors in infants are probably congenital rather than acquired. Avitaminosis A and influenza may be factors in causing metaplasia of the bronchial epithelium with later development of cancer. If the tumor is small and near the periphery it should be removed surgically. High voltage roentgen treatment may be useful for palliation when the disease is farther advanced.—*Audrey G. Morgan.*

BLACKFORD, STAIGE D., and CASEY, CARLTON J. Pleuropulmonary tularemia. *Arch. Int. Med.*, Jan., 1941, 67, 43–71.

In the thorax, as elsewhere, tularemia has been found to be primarily a necrotizing process, with foci of necrosis as the outstanding lesion. Lillie and Francis have described pulmonary lesions as basically nodular or confluent nodular in character, sometimes lobar in extent and involving one or all lobes. The pleurae may show fibrinous, fibrinocellular or fibrinocaseous exudation. These changes involve two processes: necrotization and a cellular reaction of the reticulo-endothelial-monocytic-epithelioid type.

These changes may simulate focal pneumonic lesions such as found in tuberculosis. Exudation into the pleural space may occur. Peribronchial infiltration may develop. More rarely, there may be a pneumothorax. Intrabronchial exudates may be responsible for atelectasis.

In 1935, thirty-five cases were reported from the University of Virginia Hospital. The present report pertains to 60 additional cases. Males outnumbered females approximately two to one as did also the ratio of whites to blacks. Seventeen of the group were less than twelve years of age. More than half were either farmers or housewives. Sixty-three of the 95 cases were ulceroglandular; 21, typhoid type; 7, glandular and 4, oculoglandular. Most of the infections were accounted for by rabbits but a few apparently resulted from contacts with ticks, squirrels, opossums, weasels or cats. The incubation periods varied from a few hours to two weeks; these were followed by ulcers or enlarged lymph nodes. It was because of the latter that most of the agglutination tests were performed but, in some instances, there was merely the suggestive history of contact, an unexplained fever or a chest condition of unknown cause. Diagnoses were confirmed by positive agglutination in all but one instance (in which case, it was confirmed by postmortem isolation of *B. tularensis*). The mortality rate for the series was 10.5 per cent.

The incidence of pneumonia in these infections has been variously reported as from 12.7 to 18.2 per cent. The onset of this pneumonia was usually more abrupt than that of a pneumococcic pneumonia. Physical signs were usually present within three weeks of the onset of the infection. Associated symptoms included cough, pain in the chest and dyspnea. The pulse and leukocyte count were usually low. Temperatures reached 104°F. or even higher. The duration of the illness was variable, pulmonary consolidations disappearing within ten days or persisting as long as six weeks. The mortality rate, with pneumonic involvement, is probably about 30 per cent.

Pleural fluid was obtained from 13 of the 95 patients, an incidence of 13.7 per cent. These effusions appeared to occur somewhat later after tularemic infection than in pneumonic processes. High fever was noted only in those patients who had associated pneumonia. The leukocyte count was usually normal or only slightly elevated. The amount of fluid varied from 100 to 3,650 cc.; its color usually being

yellow with little or no cloudiness. Positive agglutinations were found with this fluid as well as with blood serum. In 3 instances, inoculations of guinea pigs produced death from tularemia. Pneumothorax was rare.

Bronchitis was a common observation, it being noticeable in 19 of the 95 cases, some of which showed peribronchial roentgen evidence. Peribronchial prominence may persist for years. —*A. A. de Lorimier.*

SCHATTENBERG, H. J., and RYAN, JOHN F. Lymphangitic carcinomatosis of the lungs. *Ann. Int. Med.*, March, 1941, *14*, 1710–1721.

Lymphangitic infiltration of the pleura, peribronchial and perivascular lymphatics is not uncommon. It is frequently a secondary manifestation of carcinoma of the stomach (scirrhous type); other primary sites include: bronchus, breast, rectum, kidney, ovary, tongue, prostate and liver.

Gastric cancer is often clinically latent, having presented complaints of respiratory or circulatory embarrassment. Occasionally, with bone marrow involvement, the clinical picture may be predominantly that of a blood dyscrasia. With Krukenberg tumors, the primary clinical manifestations may be referable to the pelvis.

The case of a colored male, aged thirty-seven, is presented. The findings in the chest suggested a tuberculous bronchial pneumonia. Necropsy revealed a large infiltrated gastric tumor with extension through the pleura of the left lower lobe. The lymphatics were markedly dilated, containing neoplastic cells.

The author emphasizes the fact that this type of neoplastic extension is usually found in individuals under forty. The respiratory symptoms include progressive dyspnea—possibly cyanosis; possibly slight cough. The roentgen appearance is of a "strained design"—prominent branchings arising from the hila and developing into a finer network toward the periphery. The hilar lymph nodes may be prominent. There may be evidence of cor pulmonale. This condition must be differentiated from Ayerza's syndrome, pulmonary tuberculosis, pulmonary mycosis and blood dyscrasias.—*A. A. de Lorimier.*

SNIVELY, DANIEL, SHUMAN, HARRY, and SNIVELY, WILLIAM D. Spontaneous hemopneumothorax. *Ann. Int. Med.*, 1942, *16*, 349–356.

Spontaneous hemopneumothorax as distinguished from hemopneumothorax caused by trauma or following artificial pneumothorax is considered. In 1937 only 46 cases could be found in the literature since 1900.

The authors describe a case in a schoolboy suddenly taken with intense pain in the right side of the chest. He was a pale, asthenic youth whose health had been good, but he did have a history of having felt pain in the right chest on wrestling two months previously.

Fluoroscopic examination showed complete density of the lower two-thirds of the right lung. Thoracentesis on the right side posteriorly gave pure non-clotting blood, 450 cc. of which was removed by aspiration. There was air above the effusion. The patient was relieved of dyspnea but still felt very weak. The hemoglobin content of the aspirated blood was 65 per cent, the same as that of blood taken from the finger.

Ten days later 1,050 cc. more of non-clotting blood was removed. After that the chest roentgenogram gradually cleared up concomitantly with a decrease in the rate of sedimentation of the erythrocytes. After a little over four months the chest was completely clear, the excursions of the diaphragm normal and the patient was free of symptoms. Roentgenograms of the chest at different stages of the process are shown.

In such cases it is important to determine whether the hemorrhage is progressing. This can be accomplished by frequent blood counts on capillary blood, blood pressure examinations and serial chest roentgenograms or fluoroscopic examinations.—*Audrey G. Morgan.*

DOUB, HOWARD P., and JONES, HORACE C. Endothelioma of the pleura; clinical and roentgenologic study of three cases. *Radiology*, July, 1942, *39*, 27–32.

Three cases of endothelioma of the pleura are described and illustrated with roentgenograms. They were in women of fifty-two, thirty-three and eighteen years of age.

There has been a great deal of discussion as to whether these are true new-growths or products of inflammation. The authors are inclined to think that they originate from the endothelial cells of the lymph spaces or the lining cells of the pleura, as in most cases autopsy does not show any tumor of the lung parenchyma. They generally date from a cold or acute infection. There is generally fluid in the

pleural cavity and sometimes palpable lymph nodes, though this is not common. In the early stages the roentgenogram shows diffuse clouding with or without fluid. Later fluid appears and rapidly reaccumulates if it is removed. The flat tumor nodules on the surface of the pleura cannot be seen on roentgen examination unless the fluid is withdrawn and air introduced to produce an artificial pneumothorax. This is a valuable method of diagnosis in these cases. Diagnosis was made by this method in 2 of the cases here described and in all of them the diagnosis was verified by postmortem examination.—*Audrey G. Morgan.*

Penner, Abraham, and Druckerman, Leonard J. Segmental spasms of the esophagus and their relation to parkinsonism. *Am. J. Digest. Dis.*, Sept., 1942, *9*, 282–287.

Penner and Druckerman report 2 cases of spastic pseudodiverticulosis of the esophagus (functional esophageal diverticula of the esophagus).

One patient entered the hospital because of symptoms of complete esophageal obstruction, so severe that a gastrostomy had to be performed for alimentation. This patient was watched for twenty-two months and continued to exhibit roentgenologically typical spastic segmentation of the esophagus. Both cases occurred in aged individuals who exhibited evidences of parkinsonism. Administration of belladonna alkaloids caused in both cases complete disappearance of the spasms, and marked amelioration in the case which presented esophageal symptoms.

The clinical association of parkinsonism with the syndrome of spastic pseudodiverticulosis of the esophagus in which changes observed roentgenographically are located in that portion of the esophagus containing striated muscle, correlated with the subjective and objective improvement obtained with belladonna alkaloids, and the recurrence seen on stoppage of the treatment, lends support to the suggestion that the so-called spastic pseudodiverticulosis of the esophagus is one of the manifestations of parkinsonism.—*Franz J. Lust.*

Kaplan, S. Bernard, and Zwaifler, Nathan. Peptic ulcer of the esophagus. *Rev. Gastroenterol.*, March–April, 1942, *9*, 108–112.

Peptic ulcer of the esophagus usually occurs in the lower third of the esophagus, near the cardia, usually single, round or oval in outline when small, and may be irregular in outline and as large as 10 cm. in diameter. If the ulcer is of recent origin and superficial, the edges are clear cut and well defined and the base is red and granular. If the ulcer is of long duration, it presents a typical punched-out appearance with a funnel-shaped base similar to gastric ulcer. The ulcer may heal spontaneously with varying degrees of cicatricial contraction or stenosis, or may rupture into the peri-esophageal connective tissue, pericardium, or the right or left pleural cavity.

The symptomatology of esophageal ulcers is pain occurring directly after the act of swallowing, or even during the act of swallowing. This pain is usually a constant symptom and occurs in the epigastrium beneath the sternum and may radiate toward the back between the shoulder blades. Dysphagia is a prominent symptom; it is at first observed only on taking solid food, but later there is difficulty in taking even liquids. At the beginning there is a spasm which disappears rapidly. As the spasm increases, regurgitation follows and finally, in the stage of cicatricial stenosis, the obstruction is continuous. Vomiting is first due to spasm and later to stricture. Nutrition is undermined, the patient loses weight. hemorrhages may occur in about 40 per cent of the cases of esophageal ulcer. When hemorrhage occurs it is without nausea or effort.

The authors report the case of a boy, aged nine, who had had difficulty in swallowing and frequent periods of vomiting since birth. During the past year he would frequently awake with pain in the upper epigastrium which was relieved by sipping a little bicarbonate of soda solution. It was almost impossible for him to swallow solid food. The roentgen examination of the esophagus showed a concentric constriction at about the level of the eighth thoracic vertebra. Above this point the esophagus was partly dilated; however, the esophagus showed a certain elasticity. The direct examination revealed a small circumscribed easily bleeding ulcer. The pathological examination of a biopsy showed a gastric mucosa with erosion.—*Franz J. Lust.*

Doane, Joseph C., and Pressman, Robert. Antemortem diagnosis of tumors of the heart. *Am. J. M. Sc.*, 1942, *203*, 520–524.

Metastases of malignant tumors to the heart are not extremely rare, but until recently most

of them were diagnosed on autopsy. But in recent years 19 cases have been diagnosed before death and the authors present a 20th. A table showing the prominent features of these cases is given.

In one group of patients with heart metastases the symptoms are not suggestive. The patient may have a sudden congestive heart failure, show the picture of subacute bacterial endocarditis or may die suddenly and unexpectedly. In another group the symptoms are more definite. There are various abnormalities in the rhythm of the heart, such as auricular fibrillation, heart block, extrasystoles or paroxysmal tachycardia. One of the most significant findings is fixation of the right border of the heart shown on fluoroscopy.

Whenever a patient with a history of cancer, particularly of the adrenals, bronchi, lungs, or bones shows any of the above symptoms or an Adams-Stokes syndrome, malignant metastasis in the heart should be suspected if there is no evidence of rheumatic or arteriosclerotic heart disease, toxic conditions or a history of an old cardiac infarct.

The case described was in a woman of thirty-two in whom a diagnosis of adenocarcinoma of the cervix was made in October, 1937. Beginning November 9, 1938, a series of roentgen treatments amounting to 875 r was given.

On April 12, 1941, she was admitted to hospital complaining of cough and breathlessness, swelling and cyanosis of the face and neck and greatly dilated veins over most of the front of the thorax. The right arm was greatly swollen, the left only slightly edematous. An electrocardiogram made April 16 showed sino-auricular block. On April 19, 1,200 and 1,300 cc. turbid fluid was removed from the right and left pleura.

A diagnosis of malignant metastasis involving the superior vena cava, the right heart, the pleura and the cervical lymph nodes was made on the basis of the obstruction to the return flow of blood from the head and upper chest, the sinoauricular block and the signs of metastases to the pleura.

The patient died April 22, and autopsy showed metastases to the skin of the neck and upper chest, the superior vena cava, the pericardium and the roof of the right auricle.—*Audrey G. Morgan.*

ABDOMEN

Abels, J. C., Rekers, P. E., Binkley, G. E., Pack, G. T. and Rhoads, C. P. Metabolic studies in patients with cancer of the gastrointestinal tract. II. Hepatic dysfunction. *Ann. Int. Med.*, Feb., 1942, *16*, 221–240.

The authors have shown in previous work that patients with cancer of the gastrointestinal tract have an abnormally low vitamin A content in the plasma. This was believed to be due to the fact that the livers of these patients were unable to store or distribute the vitamin properly.

They therefore carried out further tests of liver function, the technical details of which are described. It must be remembered that liver function tests are at best crude and that the liver may be seriously damaged before it shows insufficiency in response to tests.

They tested normal individuals, patients with atrophic gastritis and leukoplakia of the mucous membrane of the mouth, patients with cancer of the gastrointestinal tract and patients from whom such cancers had been removed successfully by surgery. Tables and graphs are given showing the details of the results.

These prove that patients with cancer of the gastrointestinal tract have a much higher incidence of liver insufficiency than any of the other groups examined. This dysfunction of the liver in patients with cancer of the gastrointestinal tract is especially dangerous as they are often subjected to prolonged anesthesia and serious surgical operations which would damage even a normal liver. This makes postoperative care of these patients a serious problem. The insufficiency of the liver may cause delayed healing of the wound, stubborn anemia, hemolytic reactions to transfusion, hypoproteinemia and hypoprothrombinemia.—*Audrey G. Morgan.*

Levy, Jerome S., and Smith, Randolph T. Trichobezoar. *Am. J. Digest. Dis.*, June, 1942, *9*, 198–200.

Since the first case of trichobezoar was recorded in 1779, the authors have found 173 cases of this type reported in the literature. They report their findings in a patient whose chief complaints were pains in the stomach and a mass in the stomach for the last two years. She appeared to be mentally deficient. The patient admitted swallowing hair and snuff during her three pregnancies, but not at any other time.

On examination a mass was seen in the upper part of the abdomen extending from under the left costal margin downward toward the umbilicus and across, fading out towards the right upper quadrant. This mass seemed to be smooth, rounded, and moved on respiration. It was in the position of the stomach. On palpation it felt hard, smooth, not tender, and was of the shape of the stomach.

The roentgen examination showed, on a flat plate, a shadow of increased density in the region of and in the shape of the stomach. This was demonstrated by the barium meal as an intragastric mass freely movable within the stomach, and extending into the duodenum and probably, also, into the jejunum.

At operation, a dark green-black mass of hair was removed from the stomach. It formed a perfect cast of the stomach and was covered with the usual slimy, foul, greenish-yellow fluid. The anterior surface of the fundus of the stomach about at the level of the rib margin was adherent to the anterior abdominal wall.

When the stomach had been emptied of its hair ball, a definite ulcer of the anterior wall of the stomach which had perforated and adhered to the abdominal wall was found. There was not any infiltration or exudation in the gastric wall or in the peritoneum. This was probably the cause of the severe pain experienced at the beginning of her last pregnancy. After the hair ball had been removed and the ulcer found and examined, it was decided to incise the duodenum and remove the remainder of the hair ball which could be felt to extend into the jejunum for an inch or two. As traction produced some movement more traction was employed and the entire mass was brought back through the gastrostomy opening.

The roentgenograms and photographs of the specimen demonstrate clearly the first mass in the stomach, and the second mass which filled the duodenum and upper jejunum.

Trichobezoar of the duodenum and jejunum is extremely rare.—*Franz J. Lust.*

GRAY, HOWARD K. The diagnosis and treatment of cancer of the stomach. *Surg., Gynec. & Obst.*, Feb., 1942, *74*, 487–490.

This study is based on all the cases of malignant lesions of the stomach observed in the years 1907 to 1938 inclusive (at the Mayo Clinic). The seriousness of cancer of the stomach is emphasized when it is noted that, with the possible exception of primary cancer of the lung, the five year survival rate after the diagnosis of cancer of the stomach has been made is appreciably lower than the five year survival rate in cases in which a diagnosis of cancer of the fundus of the uterus, thyroid gland, breast, uterine cervix, or large intestine has been made.

Symptomatology. (1) In 50 per cent of the cases, the "unusual" type of symptomatologic picture of a malignant lesion of the stomach was noted in which dyspepsia was the most prominent symptom and was associated with varying degrees of decline. (2) Thirty per cent of the patients portrayed the picture of "ulcer" which was characterized by epigastric pain occurring characteristically when the stomach was empty and which was relieved by the ingestion of food or alkali. (3) 793 patients had received some type of therapy for ulcers and 81 per cent of these patients had experienced definite subjective improvement. This observation would seem to discredit the value or the advisability of the use of the response to treatment as a criterion for distinguishing a malignant lesion from a benign ulcer. (4) The fact that a patient has had symptoms referable to the stomach for a long time is no criterion upon which to exclude the presence of a malignant lesion of the stomach.

Roentgenologic Diagnosis. (1) At the Mayo Clinic, approximately 9 per cent of carcinomatous ulcers had been diagnosed as benign by a roentgenologist. (2) In 18 per cent of all cases of cancer of the stomach in which roentgenologic diagnosis was made, it was not possible to determine the exact nature of the lesion. (3) It is significant that some type of abnormal lesion of the stomach was noted in 99 per cent of cases in which a malignant lesion of the stomach was present.

The author and his associates are in agreement with Allen and Welch who studied 277 cases in which a surgical diagnosis of gastric ulcer was made. In 14 per cent of these cases, the lesion finally proved to be malignant. Operation is recommended if (1) the gastric ulcer is of short duration and the patient is more than fifty years old; (2) the ulcer is more than 2.5 cm. in diameter; (3) there is no free hydrochloric acid in the gastric contents; (4) the ulcer is situated on the greater curvature or in the prepyloric region; (5) the ulcer is chronic and is on the lesser curvature.

Treatment. In most instances, the question of operability, from the roentgenologic standpoint, depends on the situation, the extent, and

the character of the cancer. Carman divided the stomach into three zones: the operable zone, corresponding to the pars pylorica; the borderline zone, corresponding to the pars media; and the inoperable zone, corresponding to the pars cardiaca. Roentgenologic evidence alone, however, should not be sufficient evidence in occasional cases to deny the patient what benefit may be derived from surgical exploration. This is particularly true of lesions situated primarily in the fundic portion of the stomach or near the cardiac orifice as great progress has been made in the transpleural approach to these lesions.—*Mary Frances Vastine.*

GARLOCK, JOHN H. Radical surgical treatment for carcinoma of the cardiac end of the stomach. *Surg., Gynec. & Obst.*, Feb., 1942, *74*, 555–560.

During the past four years, the writer has explored 25 patients with adenocarcinoma of the cardiac end of the stomach. Nine of these were found to be operable.

Carcinoma of the cardiac end of the stomach is discussed from the standpoint of:

(1) *Anatomy and Histology.* It is important to remember that carcinomas originating in the cardia assume the histopathological features of an adenocarcinoma in contradistinction to the squamous cell tumors arising in the esophagus.

(2) *Lymph Node Spread.* In general, it may be said that adenocarcinomas arising at the cardia spread peripherally below the diaphragm toward the gastrohepatic or gastrocolic lymph nodes. Extension above the diaphragm to the nodes in the posterior mediastinum occurs not infrequently. Early metastatic involvement of the liver is a frequent finding.

(3) *Symptoms.* Cancers arising at the cardiac orifice quickly interfere with the act of deglutition and produce symptoms of lower substernal pain, especially on swallowing. However, it is important to emphasize that many of these patients may have persistent indigestion which resists medical treatment or that there may be an unexplained loss of weight. However, an analysis of these cases indicates that loss of weight does not reach the extremes frequently seen in operable squamous cell carcinomas of the esophagus.

(4) *Roentgenographic Findings.* The most common findings are obstruction of the lower esophagus, irregular filling defects at the cardia, rigidity of the lesser curvature on fluoroscopic observation, and loss of the normal mucosal pattern. It may be difficult to demonstrate any abnormality when the tumor is located on the posterior wall toward the incisura.

(5) *Treatment.* Every patient presenting the symptom of dysphagia, associated with positive findings in the roentgenograms should be subjected to esophagoscopic examination. A biopsy will indicate whether the tumor has arisen from the esophagus or the stomach. There seems to be general agreement that the operation of choice for cancer of the lower esophagus and cardiac end of the stomach is a left transthoracic, transdiaphragmatic resection with intrathoracic esophagogastrostomy.

(6) *Results.* The patient with a resectable cancer of the upper end of the stomach has a 60 per cent chance of surviving the operation and an 80 per cent probability of living more than two years.—*Mary Frances Vastine.*

PALMER, WALTER L. Peptic ulcer and gastric secretion. *Arch. Surg.*, March, 1942, *44*, 452–472.

The presence of acid gastric juice is necessary for the production of peptic ulcer. Chronic peptic ulcer has only been produced experimentally by methods which prevent the neutralization, dilution or buffering of the gastric content as it passes from the stomach. The chief protection against acid attack on the gastric mucosa seems to be the thin layer of mucus with which it is covered. If this is wiped away or dissolved ulcer develops.

Chronic peptic ulcer occurs only in persons whose gastric mucosa is capable of secreting acid gastric juice. Reports of cases occurring in persons with achlorhydria are based on mistaken evidence. No cases of peptic ulcer have been found in hundreds of cases of pernicious anemia examined.

Gastric ulcer may occur in stomachs with a low secretory response to histamine, probably as a result of poor protection of the mucosal cells. The pain in peptic ulcer is also probably caused by the action of the acid on nerve endings, the pain threshold of which is lowered by inflammation.

Such ulcers could be healed by producing complete and permanent achlorhydria. Sippy developed a method of treatment based on the use of food and alkali. Rest and milk diet is one of the oldest treatments. Various extracts that depress gastric secretion, notably extracts

of the posterior pituitary, have been used. Intensive irradiation brings about depression of gastric secretion lasting a variable time. Among 28 patients treated by Palmer and Templeton complete histamine achlorhydria developed in 35 and persisted for periods of from a few days to a few months. During the periods of achlorhydria the ulcers healed.

The indications for surgical operation are due to complications. The most frequent indication is stenosis, the most urgent acute perforation. Massive hemorrhage is best treated medically at the time of the hemorrhage, though surgical attempts may be made to tie off the bleeding artery or excise the tumor. In cases of recurrent hemorrhage subtotal resection has been performed, but even this does not ensure that the hemorrhage will not recur. No medical or surgical method except total gastrectomy results in complete and permanent achlorhydria, and therefore there is no other method of permanently curing peptic ulcer. *Audrey G. Morgan.*

WALTERS, WALTMAN. Gastric ulcer, benign or malignant. *Arch. Surg.*, March, 1942, *44*, 520–530.

The author believes that all ulcers of the stomach should be removed surgically on account of the impossibility of making a definite differentiation between benign and malignant lesions. Mistaken differentiations have been made in about 10 per cent of the cases at the Mayo Clinic on roentgen examination, and even gastroscopic examination is not free of error. Moreover, malignant lesions sometimes respond for a time to medical treatment as do benign ones. Also in some cases of gastric ulcer, benign or malignant, the ulcer so interferes with the neuromuscular activity of the stomach that a marked degree of pylorospasm is brought about, which gives the mistaken impression that the lesion is in the duodenum and benign, while in reality it is on the lesser curvature and may be malignant.

Surgical treatment has given excellent results in ulcer. With progress in surgery the danger has decreased. The mortality is now not more than 5 per cent and 89 cases were operated on at the Mayo Clinic in 1939 with a mortality of only 2.2 per cent.

The author thinks the method of choice is subtotal gastrectomy extending well beyond the lesion. In occasional cases with ulcers high on the lesser curvature or the posterior wall excision of the ulcer with gastroenterostomy has given excellent results. *Audrey G. Morgan.*

DOWDLE, EDWARD. Multiple, primary nonspecific jejunal ulcers, with chronic duodenal dilatation. *Ann. Surg.*, Sept., 1942, *116*, 348–354.

Dowdle reports a case of jejunal ulcer. The patient was fifty-nine years of age, had had occasional spells of indigestion and dyspepsia for more than ten years. Occasionally he had passed black stools. His condition had grown worse, for during the last six months, previous to hospital admission, he had vomited one to two hours following meals and had lost over 40 pounds. The roentgen examination demonstrated an obstruction of the small bowel in the region of the ligament of Treitz. This had produced almost complete obstruction with marked dilatation of the entire duodenum and stomach. Autopsy revealed that there was a shallow ulcer and another annular ulcer in the duodenum. The base and border of the ulcer was thick and fibrous. The microscopic examination showed infiltration with lymphocytes; Meissner's plexus, as well as the ganglion fibers, was distinctly swollen. There was no evidence of a neoplastic condition.

According to Dowdle, only very few cases of nonspecific ulcers have been reported in the literature. This article shows the roentgenograms and the macroscopic and microscopic findings in this case. *Frank J. Lust.*

SUSSMAN, MARCY L., and WACHTEL, EMANUEL. Granulomatous jejuno-ileitis. *Radiology*, July, 1942, *39*, 48–53.

This study is based on 23 cases of granulomatous jejuno-ileitis, which is an inflammatory disease of the small intestine. There is some question as to whether it is a primary infection or occurs secondarily in patients with deficiency disease. The average age of the patients was twenty-seven years, the extremes nine and sixty-five years. Diarrhea and abdominal pain were the outstanding symptoms. Fever, vomiting and loss of weight occurred in 25 per cent. The average duration of symptoms before admission was two years with a range of from three days to eight years. Right lower quadrant pain, vomiting and malnutrition were seen in about 40 per cent of the cases. Twenty-five per cent of the patients had perianal ab-

scess with or without fistula. There was clubbing of the fingers in 40 per cent of the cases. In 2 cases there was no glossitis; there were no other signs of avitaminosis. In only 2 cases was there a macrocytic hyperchromic anemia.

In the acute stage the mucosa is very much inflamed, edematous and thickened; in the subacute stage the mucosa is hypertrophied and granular with punched-out ulcers. In the chronic stage the wall of the affected segments of the intestine is thickened with marked submucosal fibrosis. There are normal areas between the diseased parts.

The roentgen findings follow the pathological findings closely. In the acute stage differentiation between inflammation and deficiency disease is difficult, but inflammation is indicated by an acute clinical course, involvement of the terminal ileum, the presence of segments of normal bowel separating the diseased areas, stenosis and serration suggesting ulcers. In the chronic stage there is rigidity and stenosis which sometimes causes obstruction. It is probable that many cases that have formerly been diagnosed as tuberculosis of the intestine are really cases of this disease.—*Audrey G. Morgan.*

MAYO, CHARLES W., and SCHLICKE, CARL P. Carcinoma of the colon and rectum; study of metastasis and recurrences. *Surg., Gynec. & Obst.*, Jan., 1942, *74*, 83–91.

A clinicopathological study of 334 cases of carcinoma of the colon and rectum is presented and the following conclusions are noted:

(1) Regional or distant metastasis occurred in 60.5 per cent of the cases and local invasion in 39.2 per cent. Among cases in which there was hepatic metastasis no correlation could be found between the site of the primary tumor and the site of involvement in the liver.

(2) The ability of the surgeon to detect metastasis at the time of operation was found to be good. Among cases in which death occurred shortly after resection, no metastasis was found at necropsy in 82.4 per cent of those in which none was found at operation.

(3) The success of extirpation was revealed in this same group of cases, 65 per cent of those in which there was metastasis at operation being free of it at necropsy. Residual cancer was found in only 5.3 per cent of the cases in which death occurred shortly after resection. In those cases in which the surgeon believed metastatic growth to be present in the liver, the pathologist confirmed the impression in 91.7 per cent.

(4) Independent growths are felt to be responsible for many "recurrences." Polyps were present in 34.1 per cent of the cases in this entire series, in contrast to 16.0 per cent of a control series consisting of patients without cancer of the bowel. In 14.0 per cent of the cases in which polyps occurred, malignant changes were found in the polyps. Multiple carcinomas were found in 8.4 per cent of the cases. Among cases in which resection was carried out, additional independent cancers were found at necropsy in 4.1 per cent.

(5) Whenever a carcinoma is found in any portion of the colon or rectum the presence of a second carcinoma must be carefully ruled out. After operation, the malignant potentialities of the remaining mucous membrane should be borne in mind and careful follow-up studies carried out.—*Mary Frances Vastine.*

SAILER, SEATON, and MCGANN, R. J. Lipophagic granulomatosis of the enteric tract. *Am. J. Digest. Dis.*, Feb., 1942, *9*, 55–63.

Granulomatous lesions characterized by the presence of great numbers of large mononuclear cells having an abundant cytoplasm richly interspersed with coarse fat particles, droplets of fat lying free or partially phagocytosed by multinucleated foreign body giant cells, and varying admixtures of small lymphocytes, plasma, polymorphonuclear, and fibroblastic cells, have been classified histopathologically as lipogranulomas. These reactions are predominantly local in character and confined to adipose tissue.

The authors report a case of a middle aged man who suffered from abdominal pain, weakness, loss of weight, loss of appetite, and marked epigastric discomfort after eating. In addition, there was swelling of both ankles and feet. The stools were normal in color, consistency and form. There was no diarrhea or steatorrhea.

The physical findings which were outstanding were the edema of the ankles, a very low blood pressure. A gastric analysis showed complete absence of hydrochloric acid after histamine. The glucose tolerance curve was very flat. The anemia was of hypochromic type with regeneration such as is noted in chronic blood loss.

The roentgen examination showed the esophagus and stomach within normal limits. The duodenal cap was not deformed and visualized well. Some dilatation was present in the second portion of the duodenum due apparently to hypomobility. Further on in the jejunum hypo-

mobility was conspicuous and definite puddling of the barium was noted with occasional irregular areas of spasm. The valvulae conniventes were irregular, distorted and somewhat indistinct; in a few areas they appeared thickened and unduly separated. At six hours there was still a very small amount of barium within the stomach. The greater portion occupied the lower small intestine. The colon had lost its normal haustral markings. It contained scattered portions of barium and some were present in the rectum.

The pathological diagnosis was lipophagic granulomatosis of the small intestine with massive infiltration of the regional mesentric, peripancreatic, and periaortic lymph nodes; ascites; bilateral hydrothorax; diffuse organizing pericarditis; interstitial fibrosis of pancreas (slight), and chronic atrophic gastritis.

The pathological changes were characteristically confined to the inner coats of the jejunum and ileum with massive infiltration and enlargement of the draining mesenteric and adjacent peripancreatic lymph nodes. However, there appeared to be considerable variation in the degree of small bowel involvement. In several cases reviewed the lesions were minimal or lacked entirely the characteristic features common to the mesenteric nodes. Studies on the fate of lipophagic granulomas elsewhere in the body demonstrate that spontaneous resorption of the free or engulfed fat droplets is not uncommon and marked regressions in the cellular architecture occur. Such reversible changes might conceivably have taken place in the intestine without any appreciable change in the lymph node reaction. The sensitivity of the latter to roentgen therapy is of therapeutic interest. The means by which lipophagic granulomas are formed within the small intestine and regional lymph nodes is obscure.—*Franz J. Lust.*

HUBENY, MAXIMILIAN J., MARCUS, SAMUEL M., and KAGINS, ALEX B., Calcification of the gallbladder; roentgenographic and pathologic study. *Radiology*, July, 1943, *39*, 88–91.

The pathogenesis of calcified gallbladder is not definitely known. In a large percentage of the cases, Robb says 90 per cent, there are also gallstones. It is probable that calcium carbonate stones or milky suspensions of calcium carbonate are brought about by obstruction of the cystic duct by stones, cancer or inflammation, the calcium carbonate being produced by the gallbladder wall.

On roentgen examination the gallbladder has a rigid and fixed appearance and peripheral calcification can be seen. Sometimes, however, it is hard to differentiate these calcium shadows from those of gallstones, renal and ureteral calculi, calcified mesenteric nodes, calcified echinococcus cyst, calcified fecaliths in an undescended appendix and in old people calcified deposits in the sternocostal cartilages.

Six cases are reported in which calcified gallbladder was suspected on roentgen examination. Operation was performed in 5 of the cases and in one operation was refused. The diagnosis of calcified gallbladder was verified in 3 of the cases.—*Audrey G. Morgan.*

GOLOB, MEYER, and KANTOR, JOHN L. Two cases of double gall bladder. *Am. J. Digest. Dis.*, April, 1942, *9*, 120–123.

The authors report 2 cases of double gallbladder. They review the literature and find that their cases are probably the seventh and eighth cases disclosed by cholecystography.

In both cases the dye test yielded normal sequence. The organs filled, concentrated, and emptied, in response to a fatty meal. The fundi of the gallbladder were free as demonstrated in the roentgenograms taken after a fatty meal. This suggests that they were not held together by serous covering.

The roentgenograms of the first case show the double organ especially well after the fatty meal. In the second case there was a slight time difference of appearance of the dye in the second gallbladder. After a fatty meal the concentration of the dye in both gallbladders was of the same intensity.

As the organs did not show any pathological condition, operative control was not possible.—*Franz J. Lust.*

SCHINDLER, RUDOLF, and ARNDAL, O. Gastroscopic differential diagnosis of benign and malignant ulcer of the stomach. *Arch. Surg.*, March, 1942, *44*, 473–488.

The authors believe that gastroscopic examination is the best method of differentiation between benign and malignant ulcers of the stomach. They discuss a series of 113 cases in which both gastroscopic and roentgen examinations were made. In this series 6 wrong gastroscopic diagnoses were made and in 2 cases no definite results were arrived at, while 7 wrong roentgen diagnoses were made and in 15 no definite differentiation could be made.

They give as gastroscopic signs in favor of benign ulcer: perfectly sharp edges without a surrounding wall, no infiltration of the mucosa whatever, hemorrhages and pigment spots lying in a normal mucosa, arch-shaped distortion of the angulus, hourglass fold and converging folds. Signs which are not proof of benign ulcer are: normal mucosa distant from the ulcer, uniform color of the wall and the surrounding mucosa and mucosal islands within the ulcer.

Signs which speak in favor of malignant ulcer are: limiting wall on one side and blending infiltration on the other, large tumor nodes and masses around the ulcer, irregular ridges or nodes in the ulcer floor, diffuse infiltration of the whole stomach, even without visualization of the ulcer, white, crystal-like floating material on the floor of the ulcer, location in the antrum close to the greater curvature, visible ulcer in the pylorus, bleeding of the edge, dark red color of the ulcer and pale surrounding tissue, the edge blending gradually with the surrounding mucosa, ulcerations on the wall surrounding the ulcer. Gastric signs which are not proof of malignant ulcer: large size, atrophic gastritis, infiltration of the mucosa about the ulcer, callous or edematous wall, ragged edge, bell-like elevated area, necrotic material in the floor of the ulcer, regular nodes on the floor.—*Audrey G. Morgan.*

CULVER, GORDON J., BECKER, CHARLES, and KOENIG, EDWARD C. Calcified cystic tumor of the spleen. *Radiology*, July, 1942, *39*, 62–68.

Non-parasitic cysts of the spleen are rare as compared with echinococcus cysts. Among 800 splenectomies performed at the Mayo Clinic from 1904 to 1934 only 4 cysts of the spleen were found, an incidence of 0.5 per cent.

A case of such a cyst is reported in a woman of twenty-five who came for examination for appendicitis. She had pain at the site of the appendix without associated gastrointestinal symptoms. But for about two years there had also been a good-sized mass in the left upper abdomen. Roentgen examination showed elevation of the left half of the diaphragm with a large well circumscribed area of density below it. The lower border of the shadow was rounded and calcified. There was also an area of mottled calcification inside the mass. The stomach was displaced downward and to the right and the splenic flexure was displaced downward. Except for the displacement the stomach was normal.

On operation the appendix was removed through the usual incision and then a left rectus incision was made which exposed the large cystic tumor of the spleen which on removal was found to measure 22×15×10 cm.

Tumors of the left upper quadrant may originate from the spleen, left kidney, splenic flexure, stomach, left lobe of the liver or tail of the pancreas. The best method of localizing them is by roentgen examination with barium contrast. The importance of roentgen examina- in lesions outside the gastrointestinal tract is emphasized. Roentgenograms and microphotographs of the histological findings are given.—*Audrey G. Morgan.*

GYNECOLOGY AND OBSTETRICS

SPALDING, H. C. Chondrodystrophic dwarfism in pregnancy. *Am. J. Obst. & Gynec.*, April, 1942, *43*, 720–722.

Gruggisburg distinguished between proportionate and unproportionate dwarfism or nanosoma, chondrodystrophic dwarfs being in the latter group. Kaufman states that this condition may appear in either intrauterine life or some time after birth and that the deviation from normal is chiefly in the shortness of the long bones. The soft tissues are normally developed so that the redundant skin hangs in folds. These changes are due to incomplete growth of the cartilage and early cessation of enchondral ossification. When observed at birth, this condition has been erroneously called fetal rickets. Nothing is known of its etiology or therapy. A few patients have been reported living past thirty and one even to the age of eighty. The majority, however, die in the first few weeks of life. None is taller than 140 cm. Some may be of high intelligence.

Two cases of pregnancy in chondrodystrophic nanism are reported. Both patients were colored, one was 119 cm. tall and the other, 132 cm. The blood calcium in one of the dwarfs was 7.2 mg. per 100 cc. The child of one patient showed chondrodystrophy at the age of three.—*Mary Frances Vastine.*

STEIN, IRVING F. Gynecographic aid in the diagnosis of ectopic pregnancy. *Am. J. Obst. & Gynec.*, March, 1942, *43*, 400–409.

For the diagnosis of early and especially unruptured ectopic pregnancy and for tubal mole two methods of roentgen examination are avail-

able which may be grouped under the term "gynecography": (1) hysterosalpingography which is dependent upon the use of an opaque substance; (2) pneumoroentgenography which employs a gaseous medium to produce pneumoperitoneum. These two methods can be used independently or combined with each other.

Transabdominal pneumoroentgenography may be used without danger of disturbing a pregnancy. The patient is roentgenographed in the partial knee-chest posture, the technique for which has been outlined in an earlier paper. Tubal pregnancy often appears as a dense, cone-shaped shadow, the apex arising from the uterine horn. Transabdominal pneumoperitoneum is more valuable, more informative and less harmful than hysterosalpingography. The two methods may be combined in selected cases with excellent results. For example, in cases of old quiescent tubal pathology where the diagnosis of an ectopic pregnancy must be considered, a combination of the two methods may prove of value in arriving at a diagnosis. Gynecography has a negative value in the diagnosis of ectopic pregnancy in that it may show definitely intrauterine pregnancy with normal adnexa, ovarian cyst, and/or other lesions to account for the patient's symptoms, and thus rule out the presence of ectopic pregnancy.—*Mary Frances Vastine.*

HAUGEN, JOHN A., and EHRENBERG, C. J. Diaphragmatic hernia in the newborn infant. *Am. J. Obst. & Gynec.*, March, 1942, *43*, 502–507.

Before 1935, congenital diaphragmatic hernia in the newborn infant was considered to be extremely rare and unamenable to treatment. In reality, it is not a rare finding. In a series of 5,269 autopsies on stillbirths and infants up to one year of age from the Department of Pathology at the University of Minnesota, there were 38 diaphragmatic hernias, or one in 139 autopsies in this age group. This condition can be treated surgically with more than a 60 per cent recovery. Two case reports are given.

In the first case, nothing abnormal was noted until the fourth day of life when, after a breast feeding, the infant became extremely cyanotic. The cyanosis increased in severity and a roentgenogram revealed a left diaphragmatic hernia. A laparotomy done on the ninth day revealed that the entire small intestine and spleen were in the left chest cavity. Recovery was uneventful and the infant's development has continued normally.

Cyanosis and labored grunting respirations were noted at birth in the second case. Roentgen examination revealed the diagnosis, and six hours after birth the abdomen was opened and nearly the entire gastrointestinal tract and about half of the liver was found to be in the right chest cavity.—*Mary Frances Vastine.*

BALL, ROBERT P., and GOLDEN, ROSS. A roentgenologic sign for the detection of placenta previa. *Am. J. Obst. & Gynec.*, Sept., 1941, *42*, 530–533.

The roentgen sign reported is applicable only in cases with a single fetus in cephalic presentation (about 95 per cent of pregnancies) and is based on the fact that a fetal head will dip into the pelvic inlet and occupy the midcoronal and midsagittal planes of the superior strait when the mother is standing. Any pelvic tumor mass of sufficient size can displace the fetal head from the midcoronal or midsagittal plane or prevent it from dipping below the level of the brim of the inlet.

Roentgenograms of the pelvis are taken in anteroposterior and lateral views with the patient standing. Normally, in the last trimester of gestation, the fetal head will be found in the midsagittal and midcoronal planes of the superior strait, and, even if not engaged, it will be dipping below the level of the brim of the true pelvis. The authors have accepted, for a tentative working basis, a linear displacement of one-third the diameter of the fetal head from its usual position to represent the presence of an abnormal pelvic mass.—*Mary Frances Vastine.*

BUXTON, BERTRAM H., HUNT, RUSSELL R., and POTTER, CHARLES. X-ray localization of the placenta. *Am. J. Obst. & Gynec.*, April, 1942, *43*, 610–619.

The clinical material studied consists of 126 cases of painless bleeding in the last trimester of pregnancy. At present, two separate procedures are used by the authors:

(1) The taking of right and left lateral films of the entire uterus with a technique described by Snow and Powell, which is designed to give maximum soft tissue contrast.

(2) Anteroposterior roentgenograms of the pelvis taken by a precision stereoscopic technique with 4 per cent sodium iodide in the bladder. In these films also, a modified soft tissue technique is employed.

The lateral films are taken with the patient recumbent, lying comfortably on the side. No special apparatus is necessary. By the soft tissue technique employed, the placenta has been localized in 86.1 per cent of the 108 cases where this procedure was employed. Of these, 74 were fundal on the anterior or posterior wall and 19 showed the placenta to lie wholly or in part in the lower uterine segment. In 9, no placenta was seen and in 4 there was uncertainty about the significance of the shadows.

The second method of examination is employed only in those cases where soft tissue roentgenograms fail to reveal the placenta, show it to lie partly in the lower uterine segment, or are equivocal. The purpose of these films is to demonstrate an increase in the thickness of the soft tissue space lying between the presenting part and the filled bladder. Normally this space is occupied by the bladder wall, the peritoneal reflections between the bladder and the uterus, the lower uterine segment, the membranes, and the fetal scalp. It measures from 1 to 2 cm. in thickness. In central placenta previa the interposition of the placental mass may increase the distance to 5 or 6 cm. With marginal or partial previas, there is unilateral widening either anteriorly, posteriorly, or laterally.

By coordinating the two methods outlined the placenta was localized in 97.6 per cent of the cases. Out of 17 cases of placenta previa, the diagnosis was accurately made in 16. It is not intended that roentgenologic procedures should entirely supplant vaginal examinations in suspicious bleeding cases. However, the risk of starting a serious hemorrhage in an undiagnosed case of placenta previa is very great and by detecting these cases roentgenographically, such examinations have been reduced considerably.—*Mary Frances Vastine.*

STEELE, KYLE B., and JAVERT, CARL T. Roentgenography of the obstetric pelvis; combined isometric and stereoscopic technique. *Am. J. Obst. & Gynec.*, April, 1942, *43*, 600–610.

The authors include in their article a summary which is an excellent abstract of the material discussed:

(1) A combined isometric and stereoscopic technique provides for adequate roentgenographic survey of the obstetric pelvis. Both the lateral and frontal films are taken under identical positioning of the patient.

(2) A lateral film containing an isometric scale is employed for measurement of the anteroposterior diameters of the pelvic canal and for the vertical heights of the various planes of the transverse diameters.

(3) A distorted set of scales has been developed for measuring the transverse diameters of the pelvis directly on the frontal film. The planes to be used are obtained directly from the lateral film.

(4) Only two films are essential. However, for complete examination, another frontal stereoscopic film, and a 45 degree film of the pubic arch are recommended.

(5) The precision stereoscope is preferred for study of pelvic architecture and cephalopelvic relationship and labor mechanisms as well as a check on measurements obtained from distorted images with the isometric scales.

(6) Workup of the lateral and frontal films together eliminates the location of uncertain external anatomic landmarks and measurements on the patient.

(7) Introduction of an anterior transverse diameter of the inlet, drawn between the iliopectineal eminences should assist in detecting variations in the forepelvis.

(8) Mensuration of the anteroposterior diameter, the transverse, the anterior transverse, and the posterior sagittal diameter of the inlet provides a basis for classification of pelves.

(9) A common ground has been reached for pelvic classification on the basis of mensuration and morphology.

(10) Cephalometry can be performed. Estimation of the weight of the infant from the size of the head is no longer practiced.—*Mary Frances Vastine.*

TAYLOR, ROBERT. Accessory apparatus to assist in proper posturing of the pelvic inlet. *Am. J. Obst. & Gynec.*, Jan., 1942, *43*, 140–143.

Pelvioroentgenography by the method of Caldwell and Moloy requires that films be taken with the patient supine and with the plane of the pelvic inlet parallel to the plane of the film. These workers advocated the use of a lumbosacral pad to place the pelvic inlet in the proper relation. The author noted that the head placed under the curve of the lower spine caused a great deal of discomfort to the patient so he devised an angle block with a 20 degree angle slope. The patient rests supine on this block without discomfort with a pillow underneath

the head and shoulders. Diagrams of the block are included in the article as well as roentgenograms obtained with its use.—*Mary Frances Vastine.*

THOMS, HERBERT. The clinical application of roentgen pelvimetry and a study of the results in 1,100 white women. *Am. J. Obst. & Gynec.*, Dec., 1941, *42*, 957–975.

The author recommends for routine purposes the taking of two flat films of the pelvis consisting of one view of the pelvic inlet looking down into the pelvic cavity (the inlet view) and the other depicting the lateral aspect of the pelvis. The technique employed in taking these two views is described in detail. Eight diameters which we may consider the cardinal diameters of the pelvis may be measured satisfactorily by roentgen means. These include:

(1) The anteroposterior diameter of the plane of the pelvic inlet.

(2) The transverse diameter of the plane of the pelvic inlet.

(3) The posterior sagittal diameter of the plane of the pelvic inlet.

(4) The anteroposterior diameter of the midpelvic plane.

(5) The transverse diameter of the midpelvic plane.

(6) The posterior sagittal diameter of the midpelvic plane.

(7) The widest diameter of the pelvic outlet.

(8) The pelvic height (or depth) of the outlet.

Pelvic variations as shown by the inlet conformation may be classified as follows:

(1) Dolichopellic or elongated type in which the anteroposterior diameter exceeds the transverse diameter.

(2) Mesatipellic or round type in which the anteroposterior and transverse diameters are of equal length, or the transverse diameter exceeds the anteroposterior diameter by not more than 1 cm.

(3) Brachypellic or oval type in which the transverse diameter exceeds the anteroposterior by more than 1 cm. and less than 3 cm.

(4) Platypellic or flat type in which the transverse diameter exceeds the anteroposterior 3 cm. or more.

Pelvic inlet studies were made in 1,100 white women and the distribution of the basic types was:

(1) Dolichopellic type—18.6 per cent.

(2) Mesatipellic type—41.9 per cent.

(3) Brachypellic type—32.2 per cent.

(4) Platypellic type—7.2 per cent.

The anteroposterior diameter of the inlet may be used as a criterion for a "small," "average" or "large" pelvis classification as this appears to be the most important single dimension. "Small" pelves in the various types may be designated:

(1) Dolichopellic type, anteroposterior diameter less than 12.0 cm.

(2) Mesatipellic type, anteroposterior diameter less than 11.5 cm.

(3) Brachypellic type, anteroposterior diameter less than 10.5 cm.

Labor was terminated by operative intervention 33 times (6.6 per cent) in a group of 500 patients. When the operations are separated into those which were done because of abnormal pelvic conformation, we find that the most favorable type of pelvis for successful childbearing is one whose inlet is essentially round (mesatipellic) and not the transversely oval pelvis (brachypellic or platypellic).

Studies in women of the college group as contrasted with those of the clinic group make it appear that environment during the growth period is a factor in the lessening of the number of brachy- and platypellic types of pelves found in the former group. It seems probable that, as far as variations of the pelvis are concerned, both nutritional and hormonal influences play major rôles.—*Mary Frances Vastine.*

BRILL, HAROLD M., and DANELIUS, GERHARD. Roentgen pelvimetric analysis of Walcher's position. *Am. J. Obst. & Gynec.*, Nov. 1941, *42*, 821–835.

The Walcher position of extreme body hyperextension during labor is recommended in leading textbooks of obstetrics as a useful procedure in aiding the entrance of the fetal head into a moderately narrowed pelvis. The authors question: (1) Is the Walcher position of such established efficiency as to warrant its use in spite of the great distress that it causes the laboring woman? (2) If this efficiency is conceded, is the accepted explanation correct, namely, that an increase in the length of the obstetric conjugate is produced?

Twenty patients were studied, 10 in labor and 10 during gestation. Seven of each group were roentgenographed in the comfortable dorsal position, and the remaining 3 in the moderately

flexed position. In each patient comparative measurements were made with the Walcher position. The technique carried out in those roentgenographed in the dorsal position and then in Walcher's position was as follows: The patient was placed horizontally in the dorsal position on a cart in front of an upright Potter-Bucky grid with the pelvis directly over the center of the film. The roentgen-ray beam, leaving the target horizontally, was directed transversely through the pelvis. Following the first exposure, the tube stand and Potter-Bucky grid remained unchanged, while the cart was advanced to a point where its caudal edge was approximately at the vertical center line of the Potter-Bucky grid. The patient was then placed in the Walcher position over the edge of the cart and properly centered for the second exposure. To obtain the actual measurements, a 10 cm. metal ruler was placed lengthwise over the lower abdomen of the patient directly over the midline, carefully avoiding any lateral tilting. The image of the ruler on the film indicated the exact rate of enlargement on each roentgenogram.

In their conclusions, the authors note that: (1) As compared to the comfortable dorsal position, Walcher's position does not increase the length of the obstetric conjugate in a normal profiled pelvis. (2) As compared to the comfortable dorsal position, the exaggerated lithotomy position decreases the length of the obstetric conjugate, in a normal profiled pelvis. (3) The shortening of the obstetric conjugate in the exaggerated lithotomy position led Walcher to the erroneous conclusion that the conjugate is lengthened in the position he advocated. (4) In difficult breech extraction, in a normal profiled sacrum, it is deemed advisable to change from lithotomy to Walcher's position for delivery of the shoulders and aftercoming head because the artificially shortened conjugate will thus recover its loss. (5) In flat sacrum pelves with an anterior sacral prominence, Walcher's position is contraindicated, while lithotomy position will give additional space at the inlet. (6) The two factors of increased muscular tension and altered angle of inclination may explain the reputed clinical efficiency of Walcher's position. (7) Theoretically, a device such as a low abdominal pressure-band and an elevation of the lumbar spine to a moderate lordosis may produce the advantages claimed by Walcher in a manner far less distressing to the patient.—*Mary Frances Vastine.*

D'ESOPO, D. A. The occipitoposterior position; its mechanism and treatment. *Am. J. Obst. & Gynec.*, Dec., 1941, *42*, 937–957.

A large number of pelves with correlated mechanisms of labor have been studied with the aid of the roentgen ray at the Sloane Hospital for Women. From these a random selection of 100 occipitoposteriors that terminated in easy spontaneous or low forceps deliveries and an equal number of midpelvic arrests were chosen for study.

After taking up the etiology of the occipitoposterior position, the author discusses the combined motions of descent, flexion and extension in this position. Following this, there is a consideration of the mechanism of flexion alone and its importance in the mechanism of labor. The phenomenon of rotation is next taken up and carefully discussed from all angles as it applies to the occipitoposterior position.

The considerations in the article which are of roentgenologic interest may be outlined as follows.

(1) The occipitoposterior position is caused, in the main, by adaptation of the head to an inlet possessing a narrow forepelvis and an ample anteroposterior diameter. The pelvic type which comes nearest to these specifications is the anthropoid variety.

(2) In 10 cases in the series the pelvis was so large or the baby so small in relation to the pelvis that the factors which ordinarily make it necessary for adjustment of the head to the optimum inlet diameters did not come into play and the occipitoposterior position could be largely ascribed to a matter of chance.

(3) The more narrow the pelvic brim, the more posterior is the occiput. In the true primary posteriors the occiput is at or behind the sacroiliac synchondrosis and the fetal back practically overlies the mother's spine.

(4) Large babies, small inlets, male characters, as well as the forward sacrum, convergence of the side walls, and narrow arches are distinct factors in the occipitoposterior positions that give difficulty.

(5) Two fundamentally different pelvic forms are frequently associated with posterior positions: (*a*) those in which the transverse diameters are contracted at the brim or midpelvis at both levels with compensating ampleness in the anteroposterior diameters and (*b*) those in which the anteroposterior diameters are shortened by virtue of a flat posterior segment at the

brim or a forward sacrum at the midpelvis level while the transverse diameters remain ample.

(6) Occipitoposterior positions, although physiologic and in spite of the fact that 70 per cent deliver spontaneously, cause more dystocia than the other vertex positions. Dystocia in posterior positions originates with the abnormality of the pelvis in which they occur.—*Mary Frances Vastine.*

MAYER, MAX D., NEWMAN, HERBERT, and GINZLER, ARTHUR M. Simultaneous radiologic and kinetic recording of uterine and tubal motility. *Am. J. Obst. & Gynec.*, Jan., 1942, *43*, 52–58.

A new technique for the simultaneous roentgenologic and kinetic study of uterine and tubal motility is described. Aqueous hippuran is used as the perfusing medium and a constant delivery of the medium is provided by a motor-driven syringe. Continuous pressure records are obtained through a simplified optical manometer incorporated in the system. Serial roentgenograms are synchronized to the inflation apparatus and are taken by means of a continuous strip of radiosensitive paper passed from one lead-lined chamber crossing under a radiotransparent bakelite window into a lead-lined receiving chamber.

The authors' clinical observations were:

(1) In 2 patients, no fluid could be driven past the tubes and in each case rhythmic uterine contractions were recorded while the uterus was being distended with fluid. In each case, the roentgenogram showed the typical funneling at the cornual angle and the apparent block at the beginning or in the first part of the isthmic portion of the tube.

(2) In 1 patient, a pressure tracing with rhythmic pressure waves of a range of 10 mm. Hg was obtained for a few minutes. A roentgenogram obtained while the pressure was rising revealed the left tube filled with medium with the exception of the first centimeter of the isthmic portion. This would suggest a tubal contraction in the isthmus. The waves of pressure then disappeared completely and the plateau level of insufflation pressure fell. A roentgenogram at this time showed that fluid was pouring through a completely filled right tube and the left tube was partially filled in its ampullary portion. This finding tended to support the authors' belief that the usual type of insufflation tracing with small waves of pressure is probably obtained through gas passing through a single tube rather than both tubes.

(3) In 2 patients with single tubes (the other tubes having been removed for ectopic pregnancies), gas insufflation revealed normal tracings. In these cases, rhythmic waves of pressure were observed when the fluid passed through the tubes. In both, the rises of pressure were associated with either completely empty tubes or with the medium only in the ampullary portions.

It is concluded: (1) that the method of investigation described cannot be used if both tubes are patent; (2) in insufflation of gas or fluid in human beings, the fluctuating waves of pressure are probably isthmic in origin.—*Mary Frances Vastine.*

SKELETAL SYSTEM

EVANS, WILLIAM A., JR. Recurrent fracture. *Surg., Gynec. & Obst.*, Feb., 1942, *74*, 204–219.

Seventeen detailed case reports of recurrent fractures in children are given and the following conclusions are reached:

(1) Fractures are prone to recur when a general or local disease of the bone is present, when healing has occurred with deformity, and at the site of a foreign body.

(2) A fracture may also recur at an old fracture site after healing has apparently been complete and in the absence of a significant deformity or recognizable complicating pathological process. The character of the new fracture indicates an abnormal fragility of bone at the old fracture site.

(3) Recurrent fractures of this type are most often seen in the shafts of the radius and ulna of children. Such children are apt to have poor appetites and to be of less than average weight with thin extremities. The flexible ligaments and blue sclerae which many of them show suggest a constitutional background in common with osteogenesis imperfecta.

(4) It seems desirable for children who have had fractures of the shafts of the radius and ulna to take particular care against a second injury even after the healing of the first injury appears complete and to increase their food intake for the improvement of their nutritional state.—*Mary Frances Vastine.*

McCORD, JAMES R. Osteochondritis in the stillborn. *Am. J. Obst. & Gynec.*, Oct., 1941, *42*, 667–676.

Certain skeletal changes in the fetus and newborn as seen by the roentgenogram are almost pathognomonic of congenital syphilis. McLean and Caffey are quoted when it is noted that these changes include the zigzag or saw-tooth metaphysis.

An analysis of roentgen studies on 129 Negro fetuses at all stages of gestation, the majority of which were stillborn, was made. In the study the diagnosis was made from the roentgenogram without any knowledge of the case and if the roentgenograms showed a positive or doubtful diagnosis, the babies were autopsied. The kidney, liver, spleen, lung, heart and thymus were stained by the Levaditi method and the bone lesions were reported as syphilitic only when the organisms of syphilis were found in the stained tissues. The diagnosis of syphilis as made from the roentgenogram was confirmed by the finding of the organisms of syphilis in 91.5 per cent. Ninety-four per cent of the mothers had positive serology.

The author's conclusions are:

(1) Typical saw-tooth serrations are pathognomonic of congenital syphilis.

(2) Properly done Wassermann and Kahn tests should be positive in probably 90 per cent of the mothers of syphilitic babies.

(3) It can be said, rather arbitrarily, that it is unwise to diagnose congenital syphilis by the roentgen ray in fetuses weighing less than 500 gm.—*Mary Frances Vastine.*

CLEVELAND, MATHER. A critical survey of ten years' experience with fractures of the neck of the femur. *Surg., Gynec. & Obst.*, Feb., 1942, *74*, 529–540.

This is a study of 110 fractures of the neck of the femur treated at St. Luke's Hospital over a ten year period. The author's first internal fixation of a fracture of the neck of the femur was performed in 1935. At that time the three flange nail devised by Smith-Petersen was used and since that time no fixative agent to equal it has been found. Stainless steel was the metal originally used but since 1938 vitallium has been used.

The conclusions drawn from this study are summarized as follows:

(1) *Open Reduction and Internal Fixation.* Open reduction and internal fixation of these fractures of the neck of the femur offered very little improvement in end-results over those obtained by nonoperative means.

(2) *Undisplaced Fractures.* The undisplaced fracture of the neck of the femur treated by three to four weeks' rest in bed and weight-bearing after the application of a Thomas caliper brace, gave invariably optimum results. Since the writer's nailing technique has improved, a number of these undisplaced fractures have been nailed without reduction.

(3) *Closed Reduction and Internal Fixation.* The accurate reduction by manipulation and the careful insertion of a three flange nail has yielded surprisingly good results, union of the fracture occurring in 86 per cent of the cases.

(4) *Circulatory Disturbance in the Femoral Head.* Circulatory disturbance in the femoral head, based on experience with 60 patients, has always appeared within the first year after the fracture. Seventy-five per cent of the patients with nonunion of their fractures had extensive circulatory disturbance in the femoral head. Forty-three per cent of those subjected to open reduction had extensive aseptic necrosis. Nineteen per cent of those patients who were treated by closed reduction and nailing showed some evidence of circulatory disturbance, for the most part very mild. The majority of all patients showing circulatory disturbance in the femoral head with fractures united had inadequate reduction of the fracture.

(5) *Adequate Reduction of the Fracture.* Accurate reduction is the most important single factor in securing union and avoiding circulatory disturbance in the femoral head. "Migration" of the nail is usually due to failure to properly reduce the fracture and this migration occurs almost exclusively in ununited fractures.

(6) *Nonunion.* Patients with nonunion may be greatly benefited by a trochanteric osteotomy of the femur.—*Mary Frances Vastine.*

GHORMLEY, RALPH K., POLLOCK, GEORGE A, HALL, BYRON E., and BEIZER, LAWRENCE H. Multiple myeloma. *Surg., Gynec. & Obst.*, Feb., 1942, *74*, 242–244.

In this paper, 41 cases of multiple myeloma are added to the 86 which were reported in a previous communication. This makes a total of 127 patients with multiple myeloma seen at the Mayo Clinic in the sixteen years from January, 1924, to December, 1939.

Definition. Multiple myeloma is a highly malignant tumor of the bone marrow.

Etiology. Unknown.

Incidence. Men are affected about twice as frequently as women. The disease tends to occur after the fifth decade.

Pathology. The original description was made by Dalrymple in 1846. Microscopically, the tumors are made up predominantly of cells which closely resemble plasma cells. The myeloid tissue and fat spaces seen in normal marrow are not encountered if a tumor nodule is the source of the material examined. The identity of the cells has not been established. From evidence in recent literature and in the authors' material, it seems likely that in most instances the myeloma cell is derived from reticuloendothelium.

Symptoms. There are no symptoms which are pathognomonic of the disease. Backache was the commonest symptom noted in the cases under discussion. Loss of strength and progressive weakness were the complaints which came next in frequency. Anemia, loss of weight, or occurrence of a pathological fracture were described less frequently. The presence of a palpable tumor was rare.

Albuminuria was present in about 70 per cent of the cases and Bence-Jones bodies in 60.7 per cent

Diagnosis. Biopsy or sternal aspiration; roentgenological evidence of multiple, punched-out areas of destruction in the skull, ribs, and spinal column.

Prognosis. Uniformly poor. The longest duration of the disease in the group under discussion was nine years and two months, and the shortest, four months.—*Mary Frances Vastine.*

BASSLER, ANTHONY. The intestine and chronic arthritis. *Am. J. M. Sc.*, May, 1942, *203*, 698–708.

The authors believe that chronic arthritis is caused by intestinal bacteria that enter the circulation and finally become lodged in cancellous bone or in the periarticular tissues. Streptococci are present in the intestinal tract even in healthy individuals and it is not known why some subjects resist them effectually while about 2 to 3 per cent become victims of chronic arthritis. Probably some of the ill effects are due to symbiosis of other forms of bacteria with the streptococci.

In order to test this theory the author treated 337 cases of chronic arthritis, 205 of them by diet, rectal instillations of colon bacilli which are antagonistic to streptococci and their symbiotic organisms, the oral administration of bacteriophages and in some cases autovaccines.

The diets followed the rule used in chronic biotoxic intestinal conditions. In the putrefactive or alkaline form, diets high in carbohydrates and low in protein were given and in the fermentative or acid form high protein and limited carbohydrates were given.

Among 181 cases 68.5 per cent showed marked improvement with reduction of swelling and increased usefulness of the joints and about 26.5 per cent more were improved to a lesser degree, while in the remainder of the cases there was no improvement. Most of the latter group were old cases in which there was a great deal of deformity.—*Audrey G. Morgan.*

CHASNOFF, JULIUS, FRIEDFELD, LOUIS, and TUNICK, ARTHUR M. Hyperparathyroidism in a patient with acromegaly. *Ann. Int. Med.*, Jan., 1942, *16*, 162–175.

A case is described in a woman of sixty-eight who presented the typical pictures of both von Recklinghausen's disease, due to hyperparathyroidism, and acromegaly. Photographs and roentgenograms are given showing these changes.

This patient had had a radical mastectomy in 1924 and in 1928 suffered a blow on the head. A month later she began to have severe parietal headaches and noticed that her hands were becoming large and clumsy and her features coarse.

She was admitted to Beth Israel Hospital in May, 1938, for urinary symptoms, and roentgen examination showed a large calculus almost filling the right kidney and calcareous deposits in the lower pole of the left kidney in addition to the picture of the two bone diseases.

The authors know of no previous case of the coexistence of these two diseases. The possible relationship between hypersecretion of the parathyroids which causes von Recklinghausen's disease and of the pituitary which causes acromegaly, is discussed. It is not known at present whether the patient has hyperplasia or adenoma of the pituitary. Hyperparathyroidism is generally believed to be a primary disease but in acromegaly hyperplasia of many organs, including endocrine glands, may occur. This may

be such a case of hyperparathyroidism secondary to acromegaly or the two diseases may have developed independently of each other.—*Audrey G. Morgan.*

GROSS, PAUL, and JACOX, HAROLD W. Eosinophilic granuloma and certain other reticuloendothelial hyperplasias of bone. *Am. J. M. Sc.*, May, 1942, *203*, 673–686.

A case called eosinophilic granuloma or solitary granuloma of bone was described in 1929 as a separate disease. Up to the present time 16 cases of the disease have been reported, all but three in patients less than twenty one years of age. These cases have all ended in healing, some with and some without roentgen treatment. The roentgenograms suggest osteomyelitis or tumor. This may result in needless surgery if these cases are not understood. The differential white cell count showed 4 to 11 per cent eosinophils.

The authors believe that this is not a new disease but is a form of reticuloendotheliosis closely related to the other forms of this disease, including Hand-Christian's disease, Letterer-Siwe's disease and solitary xanthoma. They describe one case each of eosinophilic granuloma, Hand-Christian's disease and Letterer-Siwe's disease and give roentgenograms and photomicrographs of the pathological tissue in support of their theory. They maintain that there are no pathologic, roentgen or other signs that can establish a differentiation between these diseases.

Eosinophilic infiltration occurs in all of them. The lesions in Hand-Christian's disease may be solitary and not generalized and it may be that cases of eosinophilic granuloma with multiple lesions will appear. Letterer-Siwe's disease is a non-lipoid reticuloendotheliosis which is generally fatal. Cases have been reported in which there were small amounts of lipoid. There are transition forms between all these reticuloendothelioses and careful autopsy studies should be made of all cases of all of them in order to establish or disprove their identity.—*Audrey G. Morgan.*

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GENERAL INFORMATION

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1. Gardner, W. J., and Frazier, C. H. Ventriculography without air injection. J. Am. M. Ass., 1929, 93, 193–194.

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PNEUMOCONIOSIS

THE STORY OF DUSTY LUNGS*

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NEW YORK, NEW YORK

A MORE scientific term for the title of this paper would have been "pneumonoultramicroscopicsilicovolcanokoniosis," which is said by the *Reader's Digest* to be the longest word in the English language. Yet, even this term is not thoroughly comprehensive.

Morbid changes in the lung caused by the inhalation of dust have become more of a social, economic, legislative and medico-legal problem than strictly a medical one.

The literature indicates that dust hazards were recognized before Christ but it has become a serious industrial problem only since the advent of technocracy or modern tools, particularly the pneumatic drill and jack hammer.

It was not until about the turn of the century that the dust hazard centered around silica, silicates and silicotuberculosis, and it was not until the second or third decade of this century that silica was considered the main offender and that most other dusts were considered innocuous. Now many observers are directing their attention mainly to the lesions that they consider are due to the inhalation of silica (SiO_2).

The more comprehensive problem of morbid changes in the lung due to the inhalation of various kinds of dust seems to have been overshadowed by the controversy concerning whether only such lesions as are caused by silica should be considered social and economic problems, or whether the morbid changes following the inhalation of other dust should be included in the legislative acts designed for the protection of persons exposed to various dust hazards.

There is even a serious controversy as to whether the silicates are capable of causing morbid changes in the lungs that are of clinical, economic and social significance, or whether the dust must be silica.

Perhaps it would be well to clarify this point by a definite statement; namely, that observers are divided into two distinct groups—one group maintains that silica is the only dust capable of causing morbid changes in the lungs of sufficient significance to become a social, economic and legislative problem; the other group maintains that dust other than silica may predominate in the lungs and that these dust particles and their accompanying morbid changes in the lungs constitute social, economic and legislative problems.

There are two investigating approaches

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that are as diametrically opposed as the above mentioned groups. The first or more conventional approach is to learn all that can be learned from the literature concerning what has been done on the subject and then to correlate those data if possible, and later attempt to prove or disprove such findings as may interest one. The other approach is to assemble all the facts that can be observed without being guided or misguided by observations and conclusions recorded by others.

The first is the conventional approach and I know of no article, certainly no recent one, that so adequately deals with the subject as the one by Pendergrass.* This essay is a masterpiece of medical literature and was presented before the Wayne County Medical Society as a memorial lecture to Dr. Preston M. Hickey. It traces the history of dusty lungs from a period just before Christ down to the present time and in proof of this he submits photographic evidence of titles or excerpts from the text of these historical documents. He quotes extensively from thirty-six different articles and emphasizes his conclusions in italics which render them readily accessible.

Much of the early literature and some of the current literature deal with morbid changes in the lungs due to dust that is not silica; one then might ask, "Why then, have some of the recent investigators picked on silica as the main or only offender?" I certainly urge all who are interested in this subject to read the article by Pendergrass, and if they feel so inclined, to compare it with the data herein assembled.

The second and less conventional approach to the subject is that of observing and recording facts regardless of whether they have been previously observed by other workers, and regardless of their apparent insignificance. Findings so observed, be they clinical, roentgenologic, or pathologic, are like sections of a jig-saw puzzle. They should be true, clear cut, well defined, so that as one begins to assemble them, they will readily dovetail with each other, without having to be forced.

Far be it from me to criticize the conventional approach which is more acceptable to most of those who are studying the subject, particularly those who have written on it. Authors take great satisfaction in having their work quoted and requoted. Intentionally we did not use the conventional method in assembling the material for the story of dusty lungs as recorded in the little blue book published by the John B. Pierce Foundation in the preliminary report on pneumoconiosis (silicosis), nor did we use it in assembling the data for the second edition of this book, which is much more comprehensive than the first and is to be profusely illustrated.

It is only since these data were assembled and described that I have gratified my curiosity as to what other observers have recorded. Again I would urge those of you who are interested in this subject to read articles by recognized authorities such as Lanza, Pancoast, Winslow, Greenburg, Gardner, Pendergrass, Drinker, Sayers, Hatch, Cummings, and many others, particularly the investigators in South Africa, who really started the serious investigation of this subject. After reading these articles you should make an attempt to correlate the observations and conclusions of these men and then set that material up and compare it with that which has been assembled for the second edition of our "Story of Dusty Lungs."

A good example of the second approach is Pancoast's investigation of the roentgenologic and clinical findings in the hard coal miners of the Pennsylvania field. His broad, comprehensive approach is indicated by the title of his first article, namely, "Effects of dust inhalation on the lungs." Indeed, it was his monumental work in this field that started the roentgenologic and medical investigation of pneumoconiosis (silicosis) in this country. About this time (1915) Lanza investigated the subject of

* Pendergrass, E. P. Some considerations concerning roentgen diagnosis of pneumoconiosis and silicosis. AM. J. ROENTGENOL. & RAD. THERAPY, 1942, *48*, 571-594.

silicotuberculosis from a statistical standpoint for the U. S. Bureau of Mines, but it was Pancoast who started the clinical and roentgenological investigations, and it was he who established roentgenology as a method of investigation in the problem of dusty lungs throughout the world.

In the late teens or early 1920's Pancoast became the leader of a group of men who were interested in this subject, but in the late 1920's and early 1930's a line of cleavage became apparent between those who followed the comprehensive approach of Pancoast, on the one hand, and those who believe that silica is the only offending dust, on the other.

At a meeting at Lake Placid in 1934 the line of cleavage split, and a definition of silicosis was adopted that rejected the "peribronchial perivascular lymph node manifestation" of Pancoast. From hearsay I understand that there was considerable controversy concerning this definition. It has been said that there were nearly as many definitions as there were men present, and that in reality the definition was a compromise. Some of those who participated in formulating the definition, or at least who sanctioned it, were not physicians, and it is evident that the majority of those present were not in sympathy with the qualified roentgenologist, as is evident by the declaration of their object, which is as follows: "A diagnosis may be required of a physician in a plant, in a public health office, in a sanitarium, or in a physician's private office. Sometimes he may have had little experience in the interpretation of roentgenograms of the chest and will be forced to lean heavily on the interpretation of the roentgenologist. The legal aspect of the problem has placed great responsibilities on the medical profession and has been a source of embarrassment. Only a physician who has examined the subject, has obtained occupational history of adequate exposure to silica dust [what is adequate?], and has before him a suitable roentgenogram of the chest [which it is admitted he is not qualified to interpret], should make a diagnosis of silicosis." The roentgenologist not in possession of these facts can merely state whether the shadows which he sees in the roentgenograms are consistent with this diagnosis. The vast majority of these cases, particularly in the early stages, have absolutely no symptoms, no physical signs and nothing on which the clinician is justified in making the diagnosis of silicosis except possibly the history of exposure to dust. Therefore the diagnosis is based fundamentally on the roentgenologic findings, and why should it not be a roentgenologist who is to interpret these findings?

They further state that, "If objective terms descriptive of the type of pathological changes could be generally adopted material progress would result. The clinician would not have to accept a diagnosis from the roentgenologist, general students of the disease would be able to correlate the findings of the various observers and more accurate definition of roentgenograms would be available for medicolegal purposes. Roentgenograms of the chest, which are notoriously difficult to reproduce for illustrations, should be described in word pictures capable of interpretation in the light of the personal knowledge of the observers." In other words, the clinician not experienced in the interpretation of roentgenograms could put his own personal opinion into the record and not subsequently be embarrassed by having to face up to prima facie evidence as shown in the roentgenogram and interpreted by one capable of such interpretation.

It is generally accepted that roentgenograms form the fundamental basis for the diagnosis of morbid changes in the lungs resulting from the inhalation of dust, regardless of what terms are employed to designate the lesion. Interpretation of roentgenograms has developed into one of the highest specialized fields of medicine, and yet it was the object of this clinical and lay groups to limit the disease by a specific definition so that the industrial physician, public health officer and the physician in his own private office, who may have had

little experience in the interpretation of roentgenograms of the chest, will not be forced to lean heavily on the experienced roentgenologist for a diagnosis of this condition, and for aiding in the solution of the social, economic and medicolegal problems, which arise therefrom.

Instead of simplifying these problems this definition has tended to make them more complicated. According to this definition, it takes ten or fifteen years of intensive exposure for one to develop silicosis. What of that group of Gauley Bridge workers, many of whom were dead in two or three years from the time of their first exposure? If it requires a history of adequate exposure to dust, what constitutes an adequate exposure both in quality and in quantity? What of the men who died without having developed typical nodulation, as defined by this definition? What of the men who had large multiple masses in the lung with very few nodules? Are all of these persons to be deprived of compensation because they were unfortunate enough to develop a manifestation of pneumoconiosis (silicosis) that failed to conform to so simplified a definition that the inexperienced industrial physicians, health officer and private physician could make the diagnosis without the aid of an experienced roentgenologist?

Since this definition was written much of the literature by those who participated in making it has been devoted to the differentiation of the unusual cases from the classical ones, as described in this definition, or they have vied with each other in coining such terms as "so-called silicosis," "pseudosilicosis" "modified silicosis," and the like. The definition previously given, and a later statement by Gardner in an elaborate article, specifically eliminates Pancoast's peribronchial perivascular lymph node manifestation of pneumoconiosis: "Insufficient exposure to silica may cause a certain amount of exaggeration of the linear shadows cast by the vascular tree and enlargements of the tracheobronchial lymph nodes. These changes are not specific of silica; they may be produced by inhaling other dusts, by infection, by heart disease. Since all these factors may be operative in some one person who worked in a silica industry, one is not justified in attributing the roentgenological appearance to silica." Let it be emphasized that without nodulation a diagnosis of silicosis is unwarranted at the present time.

After my early observations had convinced me of the value of Pancoast's work, I met him while I was demonstrating an exhibit at Atlantic City. He visited it and when I demonstrated to him our pathological findings which substantiated his roentgenological findings, he was pleased. I said to him, "What happened in 1934? Did your crew mutiny and throw you overboard?" He smiled as best he could, grasped my hand, and said, "Cole, you always could read between the lines. I'm tired. Goodbye Cole." That was the last time I ever saw him. The wonderful work, particularly on the peribonchial perivascular lymph node manifestation is still being repudiated, even by those who were closest to him.

Now as we approach the broad problem of the morbid changes in the lungs resulting from the inhalation of various types of dust and their clinical and roentgenologic manifestations by the unorthodox approach, we will do well to follow Pancoast's example, and make an intensive study of roentgenograms which are accompanied by as adequate a clinical history and physical examination as it is possible for the attending physician to give. Hundreds of such roentgenograms from many communities in this country and Canada of subjects who have been exposed to dust in many different industries are on file at the John B. Pierce Foundation, and any physician is not only permitted but is invited to study them. At the beginning of this unconventional approach, may I ask you to wipe the slate clean, if perchance you can, forget the definition of silicosis, and the controversy as to whether silica (SiO_2) is the only dust that causes morbid changes in the lungs worthy of considera-

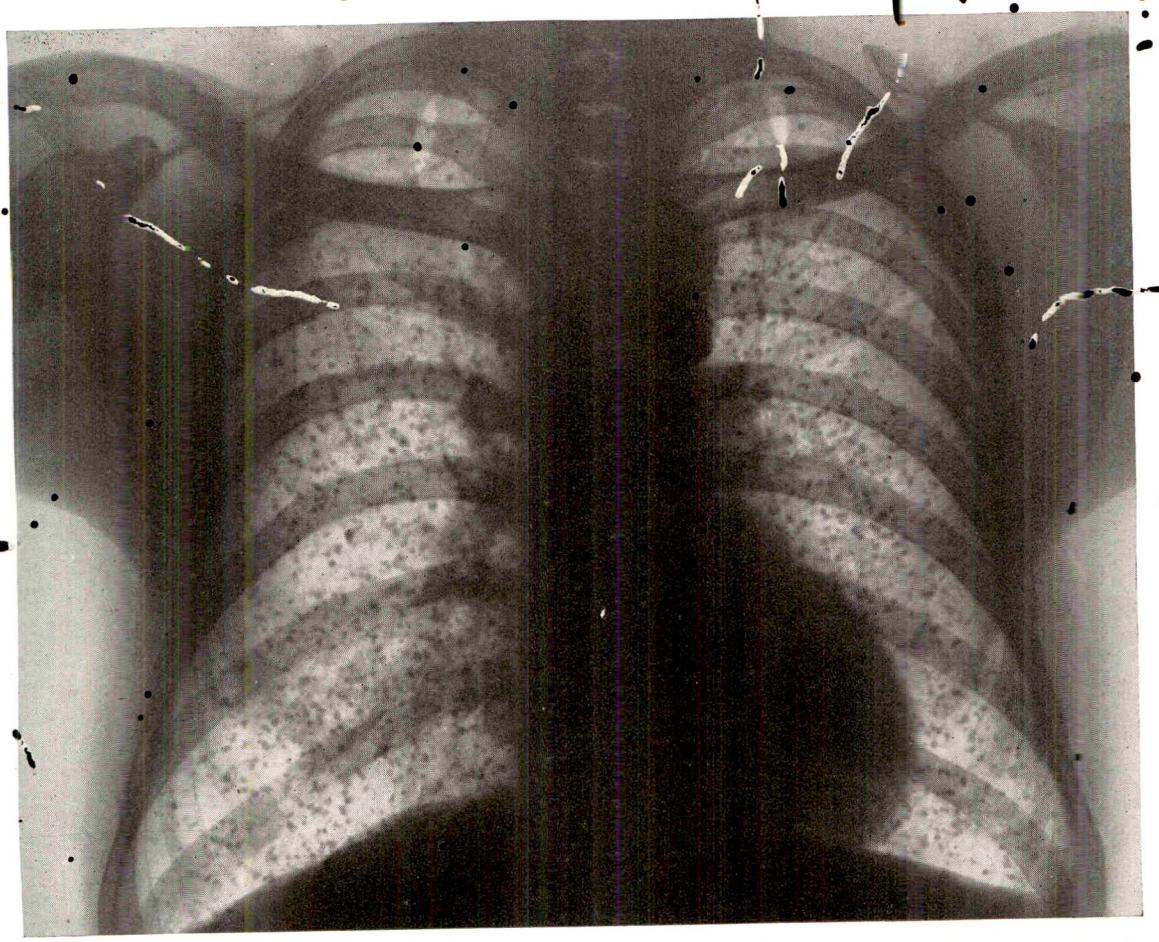


Fig. 1. (Lent by Dr. Hill, New York.) Bilateral symmetrical mottling composed of small round spots with clear cut, well defined edges. These spots are separated from one another by well ventilated lung, and bear no constant relationship to the blood vessels and bronchi as they radiate from the hilum.

Occupational and Clinical History. Patient, aged forty-eight, weight 125 lb., height 5′4″; worked since nine years of age. Until 1924 he did odd jobs and for a short period was a moulder's helper; 1924 to 1928 worked in a mixing room of an asbestos department; 1928 to 1936, as a WPA or PWA employe; 1936 to 1942 worked as a clay mixer in both wet and dry mix.

He has had coughing spells for many years which he says is better when he has good food and sleep; has no dyspnea or any other symptoms.

Social and Economic Status. He can work and wants to work.

Since the patient has inhaled a variety of dusts in different industries, who can say which dust caused these characteristic findings?

tion, or whether silicates may also cause these morbid changes in the lungs. Indeed, the problem of presenting this material would be greatly simplified if you would start at the beginning, check off paragraph by paragraph, illustration by illustration, the data herein submitted, and mark on the border of the page either approved or disapproved, or questioned, and then analyze the conclusions, and mark down whether they are rational or irrational. Do this without attempting to correlate these observations or conclusions with the controversial problems so adequately presented by Pendergrass in his conventional approach, or with the observations and conclusions of others. However, you are asked to refrain from doing this until you have followed me through to the bitter end; then you are urged to check these data and

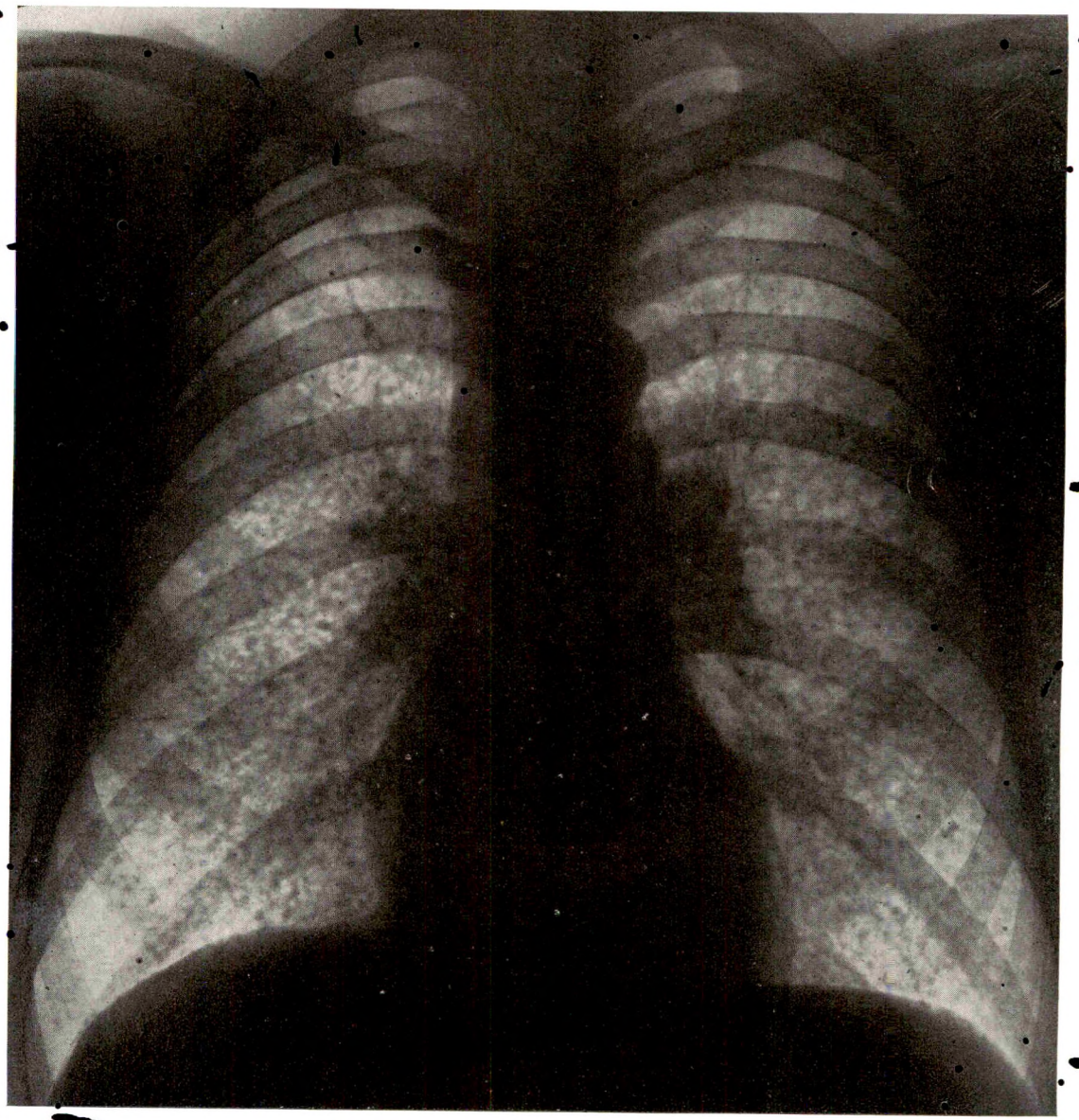


FIG. 2. (Referred by Dr. Roberto, Yonkers, N. Y.) *Roentgen Findings.* Bilateral symmetrical mottling with small irregular shaped spots that tend to join each other. The shadows are not so clear cut as those shown in Figure 1, and they bear a definite relationship to the smaller blood vessels and bronchi as they radiate toward the periphery; the shadows at the hila are moderately accentuated. There is diminished ventilation of the mid lung fields compared with the bases and apices. When these findings are compared with those of Figure 1 their differences are distinctly discernible.

Occupational and Clinical History. Male, aged thirty-five. First exposure to dust at age of sixteen; he then worked without a mask for seven years as a dry and wet driller in mines with apparently high silica content. For the last twelve years he has had very little exposure to silica dust. He has gained weight from 130 to 153 pounds since quitting underground work. He has had some dyspnea during the past seven or eight years which did not develop until four or five years after he ceased his underground exposure to silica. It does not interfere with his work, which is now not manual labor. He plays golf on flat courses but hilly courses bother him, as does swimming.

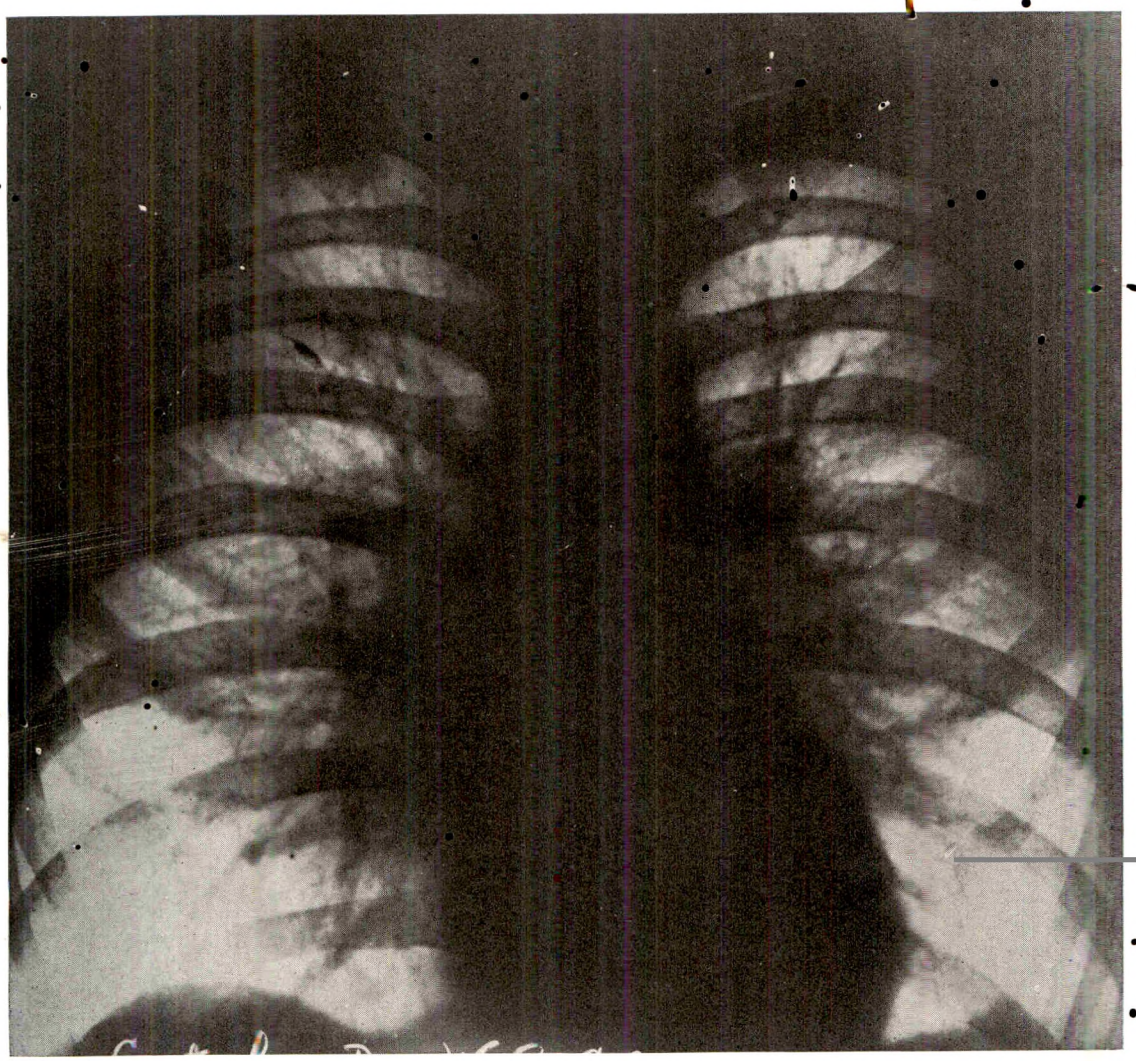


FIG. 3. *Roentgen Findings.* Very extensive accentuation of the hilar and linear markings, which is bilateral and relatively symmetrical, radiating out about as far as the middle thirds; diminished density in the mid lung fields with marked emphysema at both bases. There is little, if any, nodulation.

Occupational and Clinical History. Buffer and grinder for twenty years, using carborundum wheel. No dyspnea, cough or other symptoms.

Social and Economic Status. He could work, wanted to work, and resented being examined at routine examination lest he should be excluded from work.

compare them with those from any or all other sources.

Unquestionably roentgenograms form the basis for diagnosis of morbid changes of the lungs incident to the inhalation of dust, regardless of the dust inhaled or the term that may be applied to this lesion. If one were to observe hundreds of roentgenograms assembled from various sources, he would be impressed with the wide variety of roentgen findings. The vast majority of these roentgenograms which show morbid changes in the lungs are obtained because of routine voluntary or compulsory examination of subjects exposed to dust hazards. As indicated by the clinical history and physical examination accompanying these roentgenograms, most of the subjects have few, if any, symptoms and have very few physical signs. In some of the cases, roent-

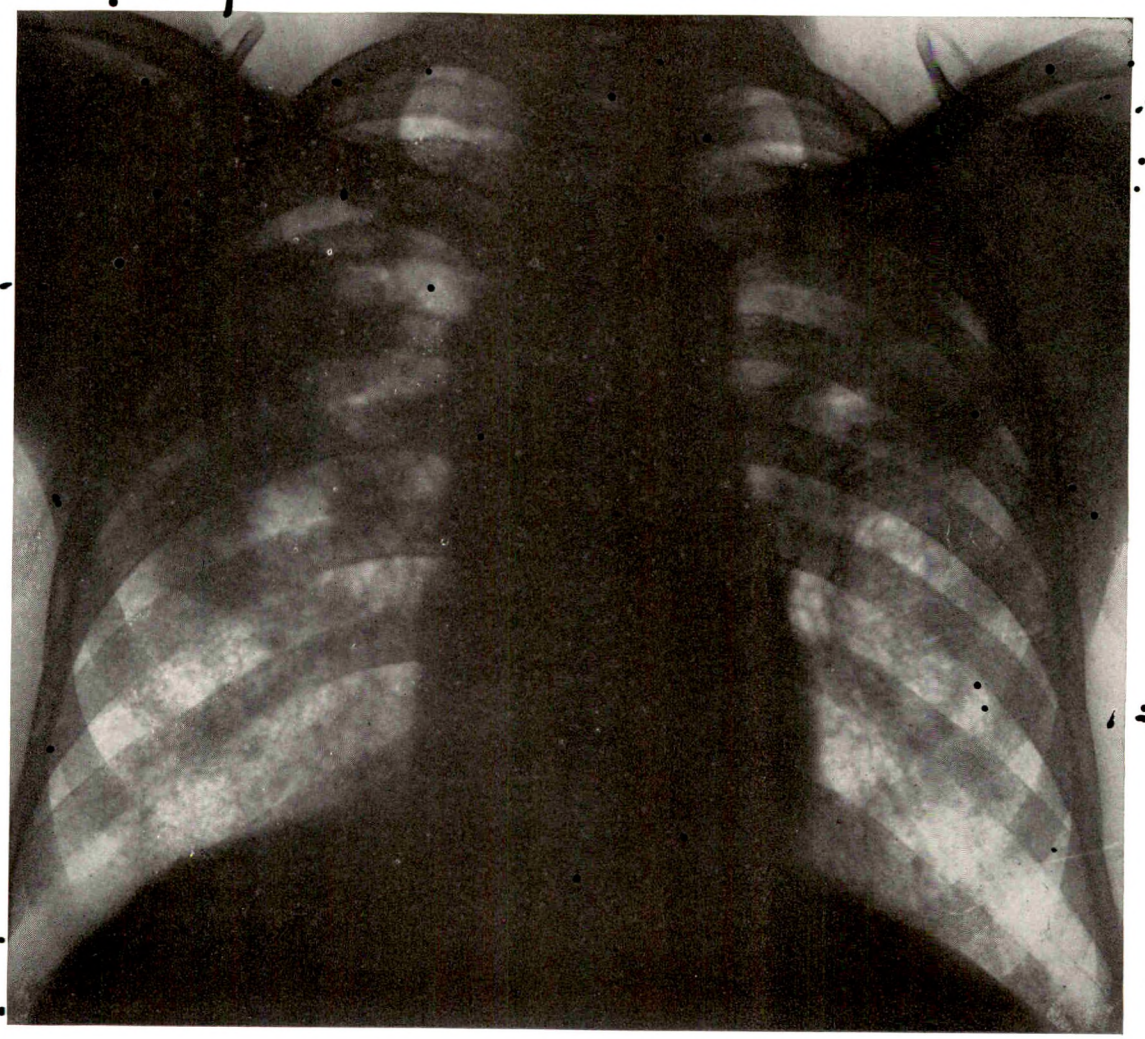


FIG. 4. (Lent by Dr. R. E. Pound, New York.) *Roentgen Findings.* Massive, dense areas of the lungs involving both upper lung fields well out toward the periphery, but the apices above the clavicles are singularly clear. The lower mid lung fields are mottled with a fine irregular pattern that bears a relationship to the smaller blood vessels and bronchi, and is very similar to the findings observed in Figure 2.

Occupational and Clinical History. Male, aged forty-five; had twelve years' exposure to iron ore mines. Light to moderate dyspnea and some cough. The massive areas of density in the upper mid lung fields is out of proportion to the history of exposure, but the fine mottled appearance in the lower lung fields is similar to that observed in iron electric welders.

genograms coincide so accurately with the definition previously quoted that they could be easily interpreted by one who has relatively little experience. But the majority of these cases differ greatly in one respect or another from the accepted definition of silicosis.

From the moment the roentgenogram is made of a subject who has been exposed to a dust hazard, if it shows some variation from the normal findings, that subject becomes a potential social, economic and industrial problem, regardless of whether the findings are slight or extensive. A few roentgenograms with both clinical history and roentgenologic findings incorporated in their legends, are selected from hundreds to reveal the great diversity of roentgen findings and the ramifications of the social and economic problem (Fig. 1–7).

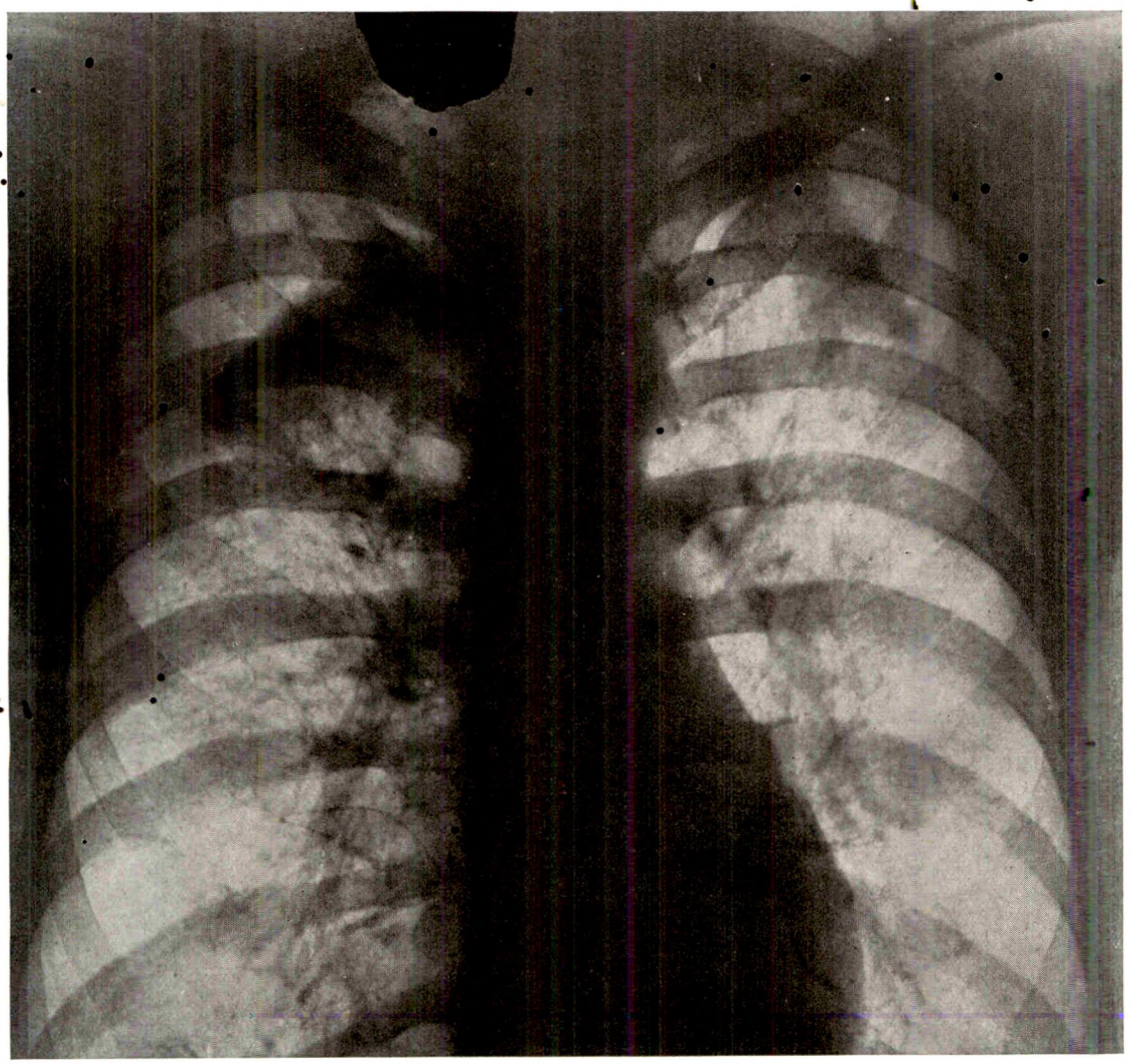


Fig. 5. (Lent by Dr. Eugene P. Pendergrass, Philadelphia.) *Roentgen Findings.* Bilateral, relatively symmetrical, smooth, homogeneous shadows involving both upper mid lung fields. "Angel wings" sign: The dense areas have sharp, clear cut upper borders but they feather out below, grading off into the normal lung density. The lung structures in the apex above this shadow are singularly normal and there is very little mottling in the lower mid lung fields. This finding is very characteristic of a dust lesion, and is due to an accumulation of dust, dust laden phagocytes, and collagen beneath the pleura at the apex of the *lower* lobes, in contradistinction to the apex of the upper lobe. This finding reminds one of rudimentary wings folded up on the back, and we have referred to it as the "angel wings" sign. No clinical history of this case is available.

An intensive study of these seven roentgenograms (Fig. 1–7), or as large a series as the reader has available, should stimulate one's curiosity as to what morbid changes in the lungs cast the great variety of roentgen shadows to which we have just referred.

Pancoast was curious concerning the morbid changes in the lungs which cast the roentgenologic shadows which he so clearly described, but evidently he could not get the cooperation of the pathologists that he desired in his own hospital, that is, the Hospital of the University of Pennsylvania Medical School, because in 1935, I searched the pathology files of this institution and

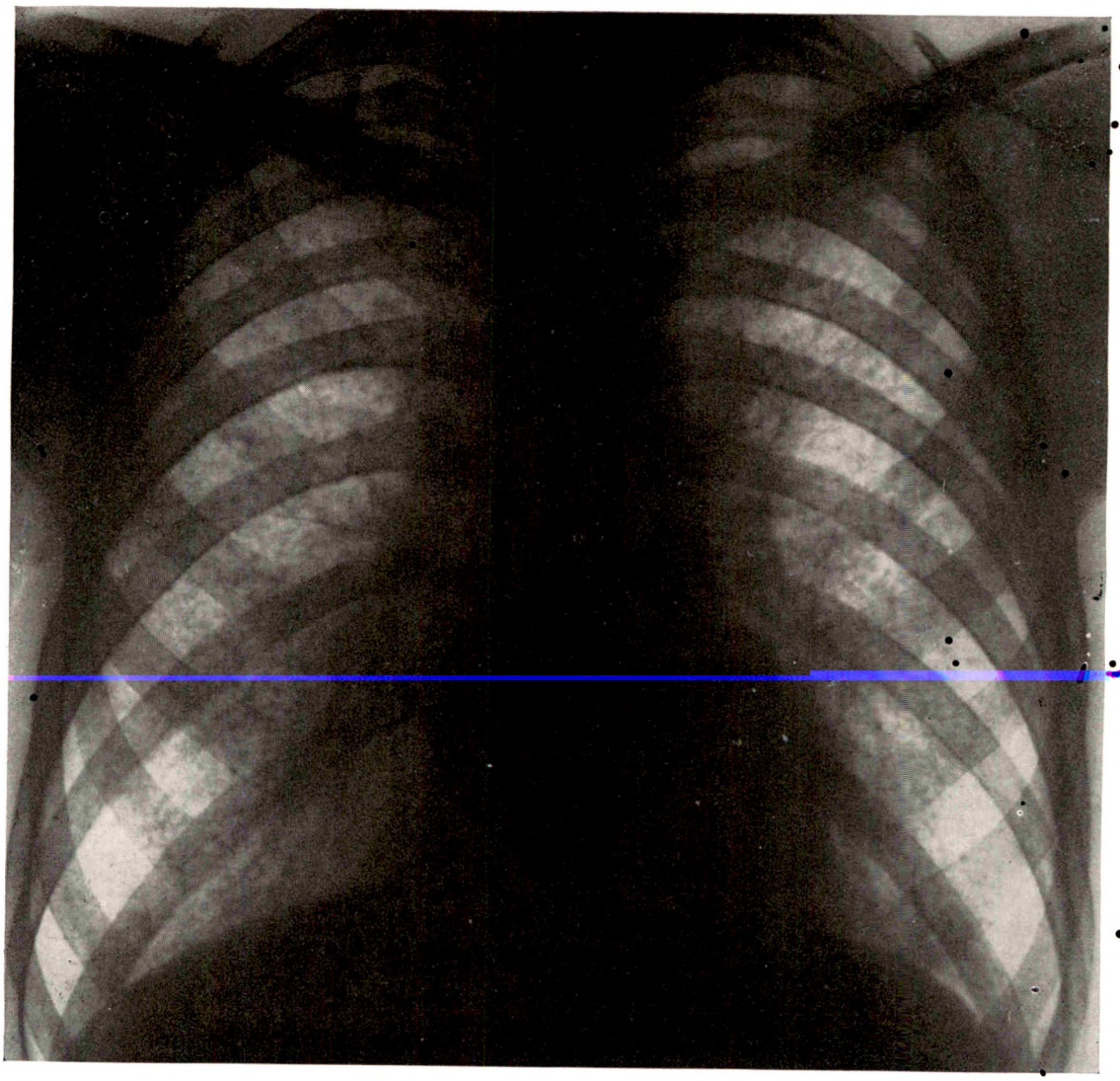


FIG. 6. (Referred by the late Dr. I. Seth Hirsch, New York.) *Roentgen Findings*. Bilateral, relatively symmetrical mottling, with very fine, indefinite spots that are almost identical with the findings observed in the case of seven years' exposure to silica as described and illustrated in Figure 2.

Occupational and Clinical History. Male, aged twenty-five; worked in a factory where his only exposure to silica was an occasional grinding of machine tools and a single exposure to emery dust which was blown into his face by a gust of wind through an open window. He has real, extreme dyspnea, clubbing of the fingers and deformity of the finger nails.

Social and Economic History. The patient sued for $75,000 and settled for $7,500. After the settlement he was intensely studied under my supervision. Slightly enlarged axillary lymph nodes revealed microscopic findings interpreted by several pathologists familiar with sarcoids as being characteristic of this disease. Other pathologists questioned this diagnosis and even doubted the existence of such a disease as a pathological entity.

This is inserted to illustrate those lesions of the lungs which resemble pneumoconiosis (silicosis) but where the clinical and pathological data do not support the roentgen findings.

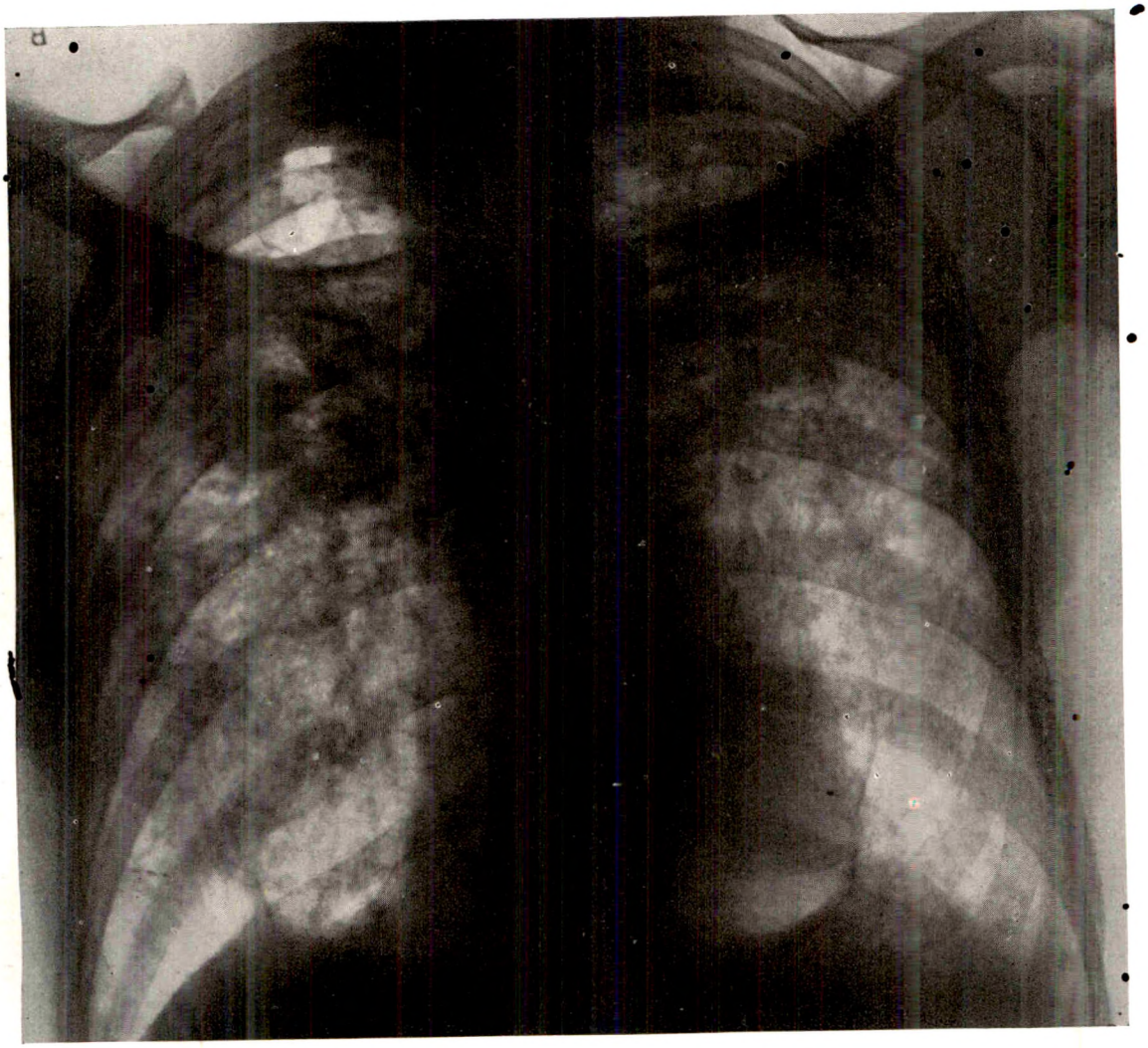


Fig. 7. (Referred by Dr. Francis Ryan, Valhalla, N. Y.) *Roentgen Findings.* Bilateral, symmetrical mottling with spots of irregular shape that are disseminated and separated from each other in the lower mid lung fields, and form relatively large dense masses in the upper mid lung fields, more advanced on the left than on the right. There is a tenting of the diaphragm on both sides, with obliteration of the costophrenic angle on the left. The outline of the heart is markedly obscured even in the region where the lung is not very dense.

Occupational and Clinical History. Male, aged sixty-six. He was literally born and brought up in a dusty atmosphere with high silica content. His father cut granite street paving blocks by hand in his own backyard in England. At the age of sixteen he became a stone cutter's apprentice, and on his first job he helped to cut a stone in a step that was just a mile above sea level. He worked constantly for fifty years as a granite cutter, without a mask and without a ventilating system except for the last four or five years. He definitely maintains that he was not short of breath during this time, and this was confirmed in part by the fact that he was able to work, and at sixty-five he stopped working only because of some Union complications. About three years ago his dyspnea came on suddenly while he was mowing his lawn. The attack was as sudden as a coronary thrombosis. He has been under my personal observation for about three years, his dyspnea gradually progressing. It has periods of exacerbation and is definitely relieved by intravenous injections of 10 cc. aminophylline. He has some cough but very little expectoration; no elevation of temperature; sputum was negative for acid-fast bacilli, and guinea pig inoculation was negative.

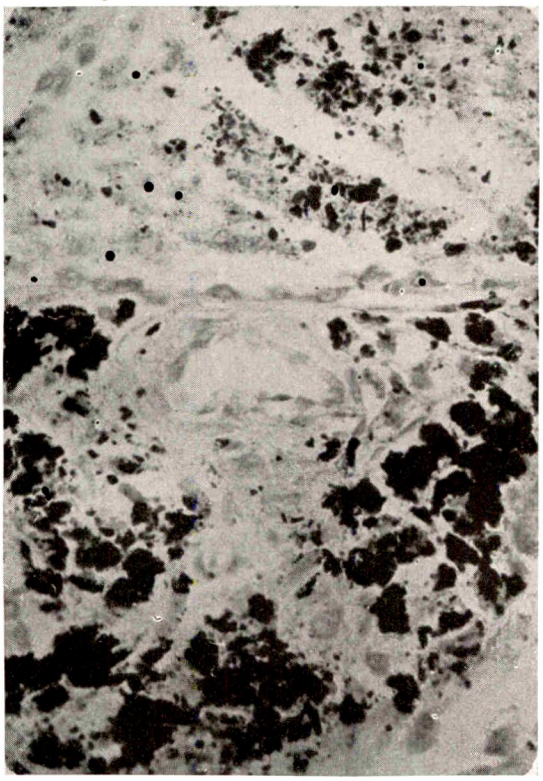


FIG. 8. The great variety of roentgen findings in the previous seven illustrations should stimulate one's curiosity concerning the microscopic characteristics of the inhaled dust and the morbid changes in the lung associated with it, as shown in this and the following illustrations. The story of dusty lungs is very briefly and concisely told in the running legends describing the illustrations.

In this illustration, opaque non-refractive airborne flecks that vary in size and shape are seen in the upper part of the field. Similar flecks may be clumped together and form large black flecks, as seen in the lower part of the field. These are not silica because (cont. in Fig. 9)

found only two microscope sections indexed under the heading of silicosis. Both of these were reported as anthracosis, which, as a matter of fact, they were. Therefore Pancoast went elsewhere to find a pathologist who could tell him what morbid changes caused the accentuated linear markings in his group of anthracite coal miners. The one he selected not only failed to identify these morbid changes which cause the peribronchial perivascular lymph node manifestation which he had observed, but he caused Pancoast's crowning achievement of a long and useful career to be repudiated.

Even at the present day Pendergrass recognizes the need for cooperation with various specialists. In his article in the November, 1942, issue of this JOURNAL (page 577) he states, "An ideal program for the diagnosis of pneumoconiosis in the living would include a study of a group, the members of which would be a general physician, a rhinologist, a bronchologist, a clinical pathologist, a specialist in tuberculosis, an engineer interested in industrial hygiene and a general roentgenologist Such a program is too ambitious for practical purposes. . . ."

The roentgenologist is surely in need of a pathologist to aid him in the interpretation of his roentgen findings in terms of pathology, unless he chooses to do this himself.

For the roentgenologist who may be curious to know the morphological characteristics of the inhaled dust and of the morbid tissues in the lungs of those who have inhaled quantities of dust and who are desirous of examining microscope sections of lungs of such cases, or who may be fortunate enough to have the cooperation of a pathologist, an attempt will be made to describe and illustrate gross, macroscopic and microscopic characteristics of these lesions.

A casual macroscopic and microscopic examination of large microscope sections of the lungs of subjects who have been exposed to various dust hazards reveals that the morbid tissue is composed of: (a) deposits of dust, some free in the alveoli, others clumped together in phagocytes either in the alveoli or in the stroma of the lung; (b) masses of collagen laid down in various patterns; (c) cell proliferation; some fixed, some wandering phagocytes; (d) air cysts, caused by the dilatation of the terminal air passages and alveoli; and (e) intravascular deposits of debris and young and aging connective tissue cells.

A more intensive study of these microscope sections leads one to consider these

findings under separate headings: (1) lung dust; (2) phagocytosis; (3) collagen versus connective tissue; (4) six types of pneumoconiosis, and (5) vascular changes.

PNEUMOCONIA (LUNG DUST)

The term "pneumoconia" is not conventional, but there is ample precedence for its use.

Man inhales dust with every breath he takes from the cradle to the grave, sometimes little, sometimes much. Some of this dust is presumably caught on the baffle plates in the upper respiratory tracts; some on the cilia of the trachea and the larger bronchi, and wafted back; whereas other particles of dust are deposited in the terminal air passages and alveoli. Even some of the dust that is deposited in the alveoli is undoubtedly coughed up. The reason for concluding this is the manner in which iodized oil, which has reached the terminal air passages and alveoli, is evacuated from these regions within a very short time.

Such dust particles as may be retained within the lung can be observed microscopically either by the conventional light field method of examination, or by fields rendered dark by cross polarization.* Some

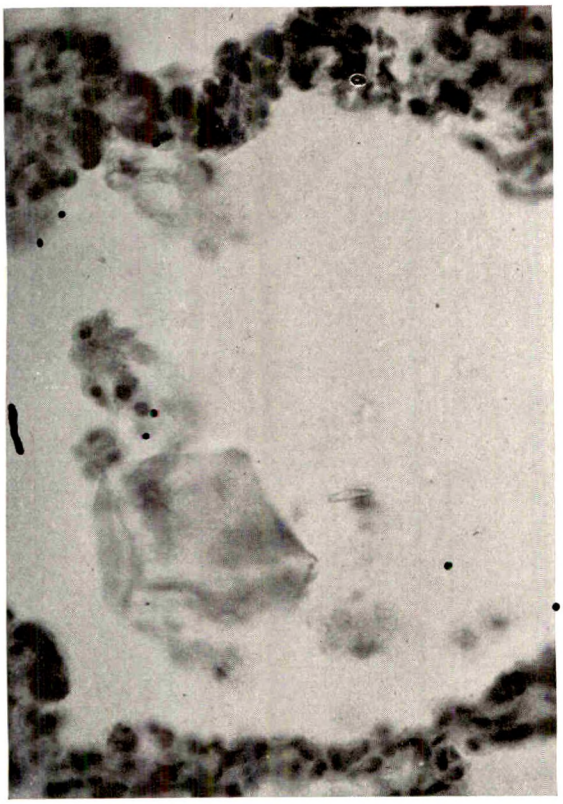


Fig. 9. Silica particles are transparent; they are clear as crystal; only a very large one that looks like an ice cube is visible, in the light field, but when (cont. in Fig. 10)

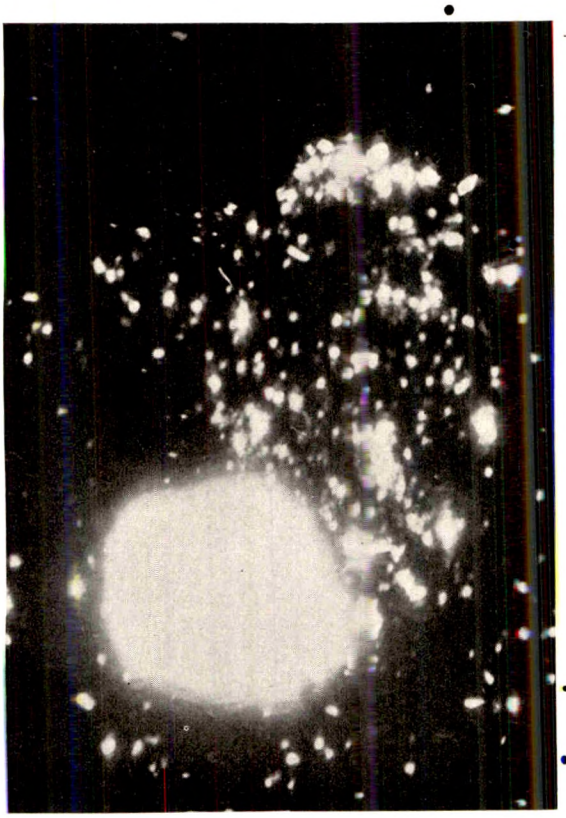


Fig. 10. The same silica particles are viewed in a field rendered dark by cross polarization, they become exceedingly brilliant, reminding one of the moon and stars, but "all is not silica that glitters."

* Cross polarization is a procedure familiar to petrologists but perhaps not to all pathologists, or at least not routinely used by them. The procedure consists of inserting a pair of polaroids or Nikol prisms, one above and the other below the microscopic section. When these polaroids or prisms are parallel, the field appears as the conventional light field, but when they are rotated at right angles to each other, the field is rendered dark and transparent dust particles that have the properties of refraction, refract the light so that each dust particle appears as a brilliant object in a dark field. This procedure is very important in the study of dust particles, and is particularly valuable in the study of such particles lodged in the lungs. It is fully described in our complete text in a chapter devoted to tecanic.

dust is air borne, some is blood borne, and some may even be of blood origin. Some of the dust particles are opaque, some translucent, and some transparent. Opaque air borne dust particles, as observed in the conventional light field, are often black or reddish brown, or translucent like a shell (Fig. 8). They differ greatly in size and shape, whereas opaque blood borne dust particles are small, uniform in size and relatively round. Other particles of air borne dust are transparent (Fig. 9); they are as clear as glass and are barely seen by the conventional light field method of examination. One would scarcely suspect their presence when viewed with either the low or high power magnification. Only by observing Burle's line around these flecks can one determine their size and shape. But when these transparent particles are viewed in a field rendered dark by cross polarization they appear as brilliant as the stars in the heavens on a clear moonless night (Fig. 10). If one is not familiar with these findings, such a microscopic field is quite startling.

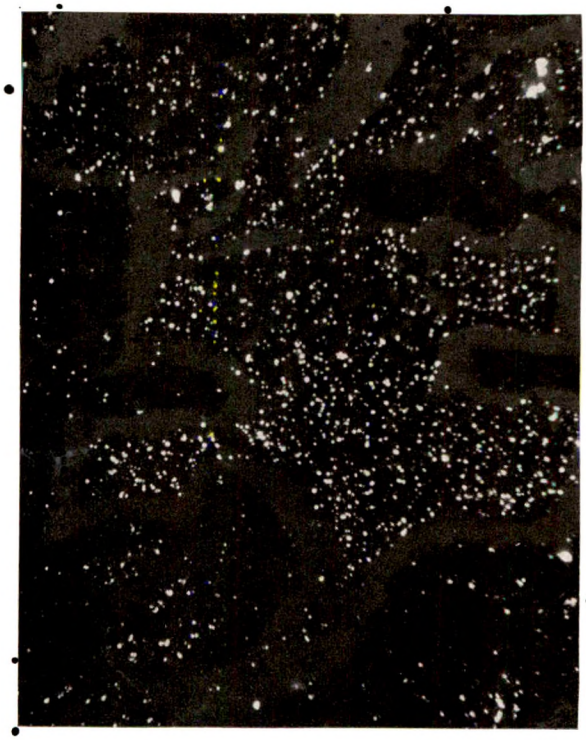


FIG. 11

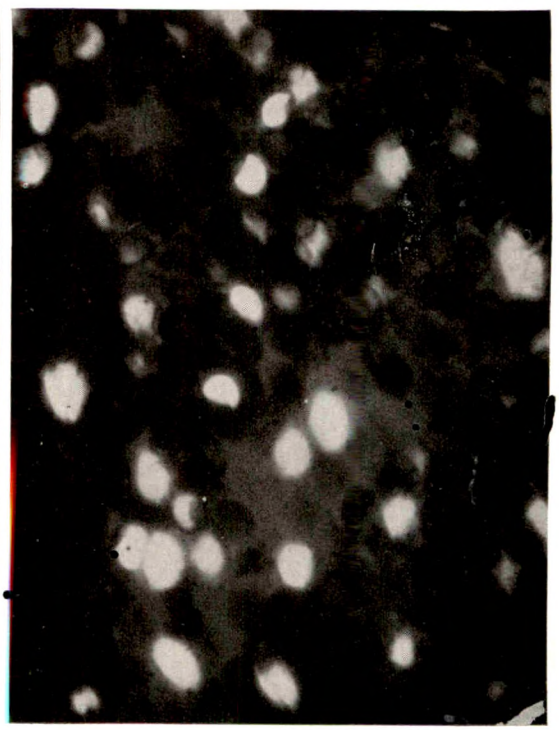


FIG. 12

FIG. 11. Hemoconia in a field rendered dark by cross polarization is as brilliant as silica, but they are small and uniform in size as compared with two or three silica crystals in the upper right hand corner.

FIG. 12. Red blood corpuscles under certain conditions may become birefractive and appear as brilliant spots in a field rendered dark by cross polarization. Such red blood corpuscles can be broken down by heat into four, six or eight fragments, and these are not distinguishable from hemoconia. These correspond in size and shape with the red blood corpuscles except for the fact that some of the fragments have a piece bitten out, so that they resemble a partial eclipse of the moon.

These birefractive particles that are brilliant in a field rendered dark by cross polarization may be silica (SiO_2) or the silicates, but "all is not silica that glitters." Indeed, all that glitters is not even air borne dust. Microscopic fields rendered dark by cross polarization may show quantities of hemokonia that so closely resemble silica that it

may be mistaken for it (Fig. 11).

Hemoconia is defined in Gould's *Medical Dictionary* as follows: "Minute, colorless, highly refractive, spheroidal or dumbbell shaped bodies, constantly present in normal and pathological blood. They are not more than 1 μ in diameter, and present active but limited molecular motility, not true ameboid motion." The distinction between small individual particles of silica or silicates and hemoconia is one of the problems of pneumoconiotic pathology, and is discussed in great detail in our complete text.

Sources of Hemoconia. The source of hemoconia is problematic. Red blood corpuscles under certain conditions may be as brilliant as silica in a field rendered dark by cross polarization. These red blood corpuscles, however, are relatively uniform in size, often with a piece bitten out of the side, so that they remind one of a partial eclipse of the moon (Fig. 12), and they are broken down into fragments by heat, whereas silica is not. These broken down fragments are not distinguishable from hemoconia. This subject has been discussed in the text of our book, perhaps to a greater extent than the subject deserves.

In our complete text the characteristics of these transparent, birefractive flecks have also been discussed in detail, but herein they are only mentioned: (*a*) relatively flat crystals have a sudden complete period of extinction as the microscopic field is rotated; (*b*) in multiple granular particles part of the dust particle disappears in one stage of rotation of the field, whereas another part becomes brilliant; (*c*) inclusions, that is black flecks within the transparent crystal; (*d*) lines of cleavage on the surface of the crystal, and (*e*) various shapes of the fragments of the crystal are all microscopic characteristics of transparent birefractive particles that may be either silica or silicates, or hemoconia.

The manner in which these opaque, nonrefractive and transparent birefractive dust particles are deposited in various regions and structures of the lung are illustrated by a score or more of photomicrographs in our complete text, but only four are used to illustrate this text. Hundreds of photomicrographs of the lungs of subjects who have been exposed to intensive dust hazards are on file in the laboratory of the John B. Pierce Foundation. The original microscope sections from which these kodachrome photomicrographs have been made are also on file in the laboratory, and the region in the microscopic field from which these kodachromes are made are recorded on each kodachrome, so that anyone, be he critical or otherwise, is welcome to come to the laboratory, bring his own petrographic microscope and check the observations herein recorded and evaluate my conclusions.

PHAGOCYTOSIS

We have guessed how the air borne dust enters the lung and how some of it has been coughed up; now let us see what happens to that part of the dust that temporarily remains deposited in the terminal air passages and alveoli.

Dust of air borne and blood borne origin and also bacteria deposited within the alveoli seem to stimulate phagocytes to emerge from the blood stream and engulf dust and other organic substances lodged within the alveoli. Many of you have seen microscopically the circulation of blood either in the window of a rabbit's ear or in the omentum of one of the lower animals, as it is spread out over the microscopic field. Those who have not seen the original circulation of the blood may have seen motion pictures revealing the circulation. As will be recalled, the blood passes through the capillaries not as a constant stream, but in fits and starts, in unison with the heart beat. Phagocytes circulating in the blood stream may first cling to the inner wall of the capillaries and then emerge through chinks in the capillary walls. They then wander around in the adjacent structures and later they either wander off into the lymph stream or reenter the blood stream through other chinks in the alveolar wall.

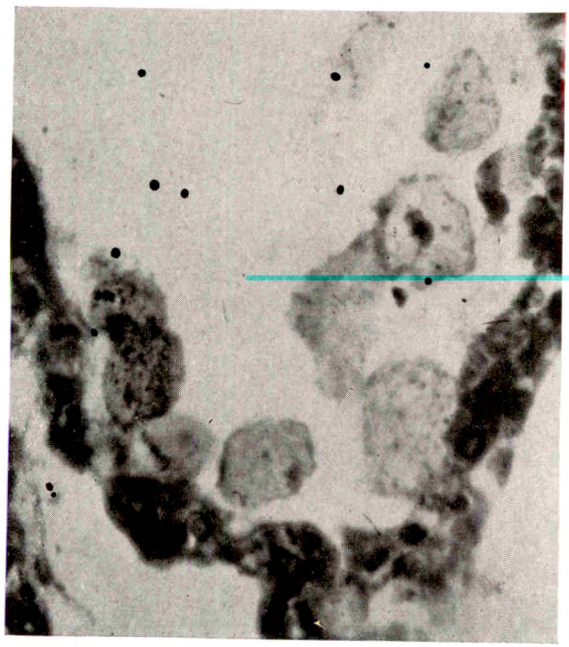


FIG. 13. Phagocytes emerge from the walls of the alveoli and mop up dust, sometimes little, as seen in the upper right field, sometimes much, as in the lower left field. This opaque dust is not silica, but (cont. in Fig. 14)

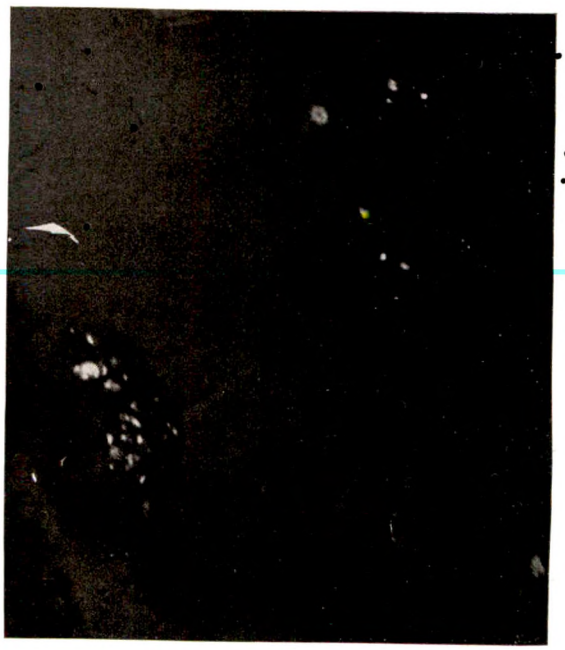


FIG. 14. Silica and hemoconia are observed when the same field is rendered dark by cross polarization; they appear as white spots on a black background.

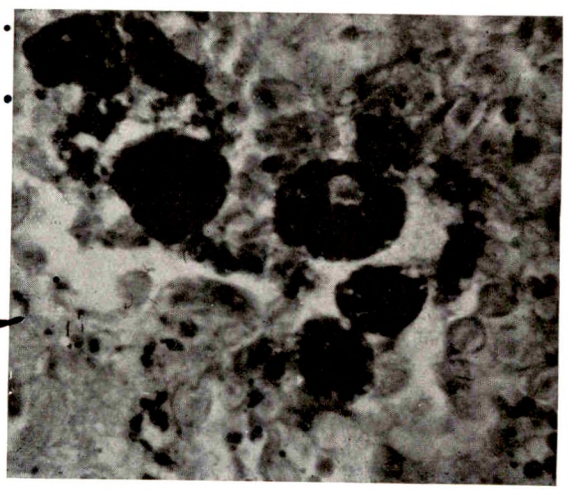


FIG. 15. If a phagocyte mops up only black dust, which is not silica, it becomes a "black phagocyte"; sometimes it takes up so much that its nucleus is obscured, or the nucleus may appear as a "red eye," as shown to the right.



FIG. 16. If a phagocyte mops up a preponderance of birefractive particles, silica, the silicates or hemoconia, it becomes a refractive phagocyte; when viewed in a field rendered dark by cross polarization the nucleus may be obscured and the phagocyte killed by its overload, or (cont. in Fig. 17)

I do not know of this experiment having been done on the lungs of humans or even on the lungs of lower animals, and it is hard for me to conceive how it could be accomplished, but one may reasonably conclude that this physiologic process known to occur in the omentum and in the rabbit's ear also occurs in the lungs of lower animals, or even in man. An intensive study of the phagocytes observed in the microscope sections of these dusty lungs seems to confirm the physiologic phenomenon just recorded. In these large microscope sections some phagocytes, as observed by the conventional light field method, contain opaque, black, brown or translucent particles. It must be borne in mind that these particles are not silica because silica and even the silicates are relatively transparent and are scarcely visible in the conventional light field.

If we now study some of the phagocytes that have recently emerged from an alveolar wall, it will be seen that some are relatively free of these opaque dust particles. Others contain a goodly number, whereas still other phagocytes in the same alveolus may be literally filled with these opaque particles that are not silica (Fig. 13), because silica particles are so transparent that they are not readily visible in the conventional light field. Only by a careful scrutiny can one see the Burle line surrounding the transparent dust particles.

When the same field is rendered dark by cross polarization, many transparent birefractive particles that were not visible in the light field become brilliant in the dark field (Fig. 14). Some of these are silica, some are silicates, and others are hemokonia.

If a phagocyte ingests only black flecks, which are not silica, it becomes a "black phagocyte" (Fig. 15). Its nucleus may be visible and appear as a "red eye," or its nucleus may be obscured by its overdistention with black particles. On the other hand, the phagocyte may take up mostly birefractive particles. It then becomes a birefractive phagocyte (Fig. 16), as observed by cross polarization, or "white phagocyte", in contradistinction to the "black phagocyte." Some of the phagocytes are apparently killed by their overload of transparent, birefractive particles. As the organic material within the dead phagocyte is absorbed, the refractive particles are redistributed on the lining membrane of the alveolus; that is, if the phagocyte is still retained within the alveolus when it dies.

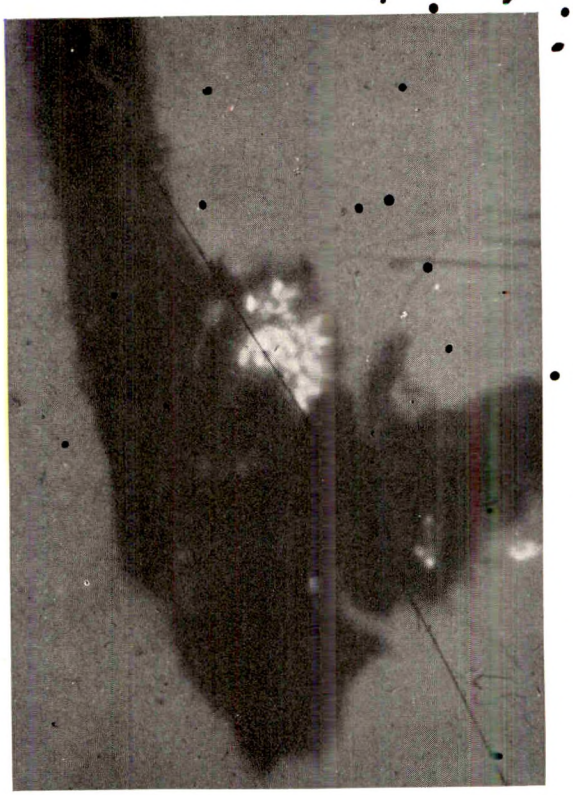


Fig. 17. A viable phagocyte may reënter the alveolar wall, the nucleus entering first, drawing its load of crystals, as Santa Claus does his pack.

Phagocytes not killed by their moderate load of either opaque, nonrefractive or transparent birefractive flecks are observed either within the alveolus or in the stroma of the lungs. In such phagocytes the living nucleus appears as a "red eye" and these living phagocytes encompass air borne or blood borne dust, hemocomia and other organic material that may have been deposited in the alveoli, and they transport them either along the lymph channels toward the hilum or toward the periphery of

the lung or they may congregate or aggregate in adjacent alveoli.

Such living phagocytes may reënter the wall of the alveolus, the nucleus first entering through a small chink in the alveolar wall, drawing its cell packed body through after it (Fig. 17). Many kodachrome micrographs and microscope sections from which these observations were made are, as we have said before, available for students and critics alike.

The manner in which these dust laden phagocytes congregate in small, irregularly spheroidal masses in adjacent alveoli is of great importance, but it will be considered in a section devoted specifically to the pathology of the dust nodule and the dust tubercle.

COLLAGEN VERSUS CONNECTIVE TISSUE

The dense areas in the lungs which cause roentgen shadows in cases of silicosis or pneumoconiosis have hitherto been considered to be connective tissue. But, as a matter of fact, our observations have indicated that connective tissue is singularly absent in the parenchyma and stroma of the lung, even in typical cases of silicosis. These deposits in the lung be they (*a*) masses, (*b*) nodules, or (*c*) radiating sheaths, are caused by dust, dust laden phagocytes and collagen, either in the alveoli or in the stroma of the lung.

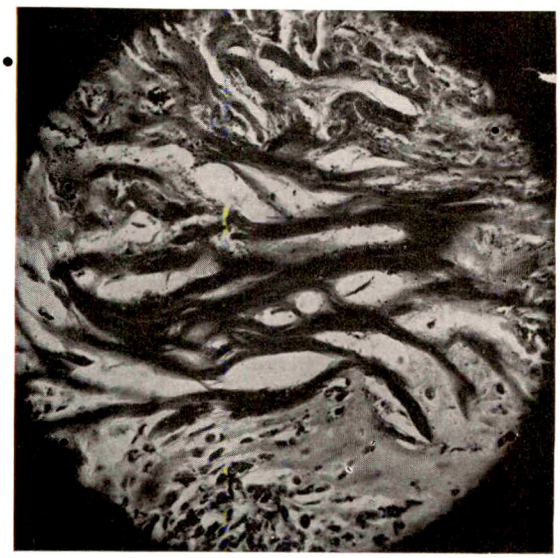


FIG. 18. Collagen, *not connective tissue*, is one of the principal factors in pneumoconiotic (silicotic) pathology. It is laid down in various patterns relatively free of nuclei, and it is easily differentiated from (cont. in Fig. 19)

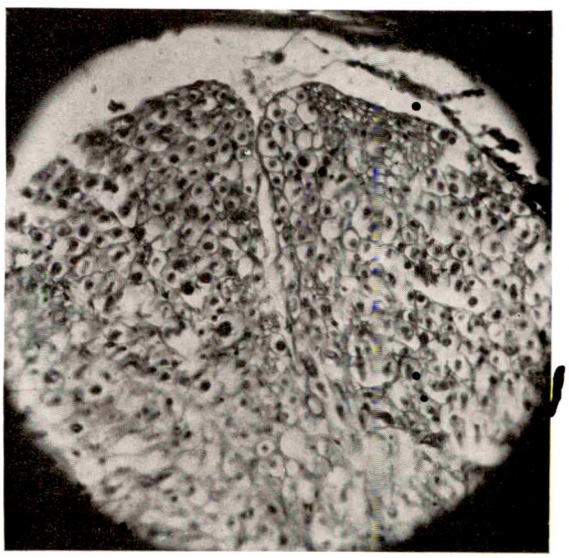


FIG. 19. Reparative connective tissue which in its early stage is characterized by cells with large, clear bodies and large nuclei; as the connective tissue ages, the cells become fusiform and eventually very long, with small oval nuclei. Collagen acts as a filler in the chinks between the cells.

Collagen in sections stained with hematoxylin and eosin is inconspicuous and difficult to differentiate from other structures, particularly smooth muscle fibers, but when collagen is stained with Masson's trichrome stain, it is easily recognized and is in great contrast with the smooth muscle tissue, which is stained red. The collagen (Fig. 18) is stained blue.

Collagen as observed microscopically in cases of pneumoconiosis (silicosis) is laid down in various patterns which have been considered under four headings in the book published by the John B. Pierce Foundation.



FIG. 20. Accurate tracings of the pulmonary artery as observed in a vasculogram, injected by Dr. Robb. The dark arteries supply the third lobe. The bronchi and veins run about parallel with the arteries. The blood vessel markings in the following schematic drawings are less accurate.

Six schematic drawings presenting my conception of the combined roentgenologic and pathologic findings of the six types of the disease are presented for comparison.

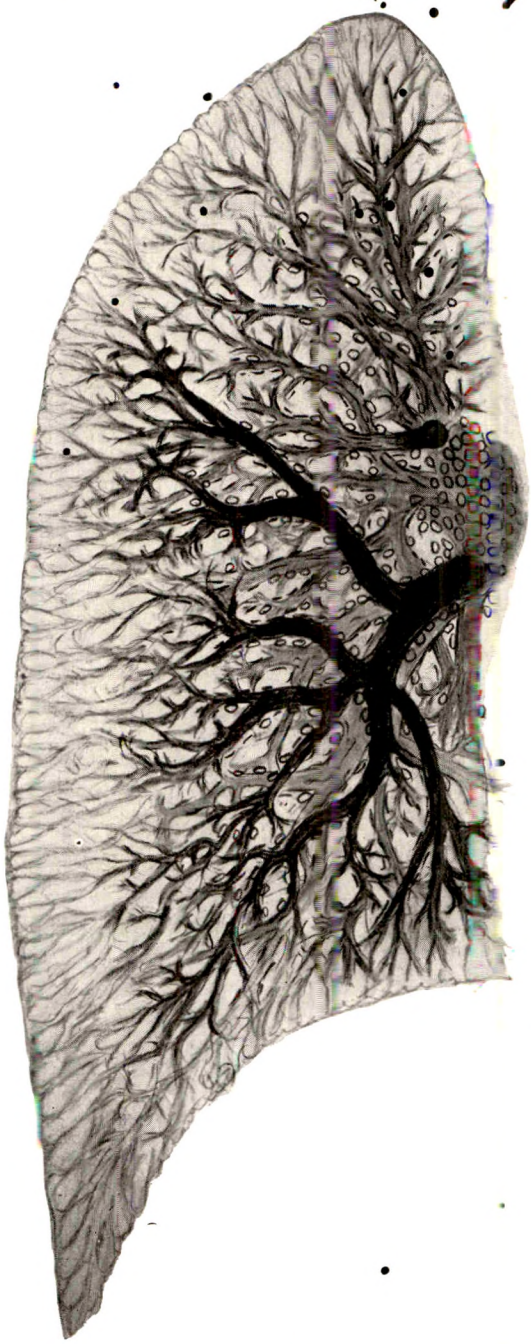


FIG. 21. *Peribronchial perivascular lymph node manifestation of Pancoast* is evicenced by accentuated hilar and linear markings that are due to dust, dust laden phagocytes and collagen deposits, laid down in laminae as sheaths surrounding the blood vessels and bronchi. The dust laden phagocytes are indicated by circles and the collagen by dashes. These findings are definite manifestations of pneumoconiosis, but often the lesion is of little clinical significance in the early stage, or even in the moderately advanced stages.

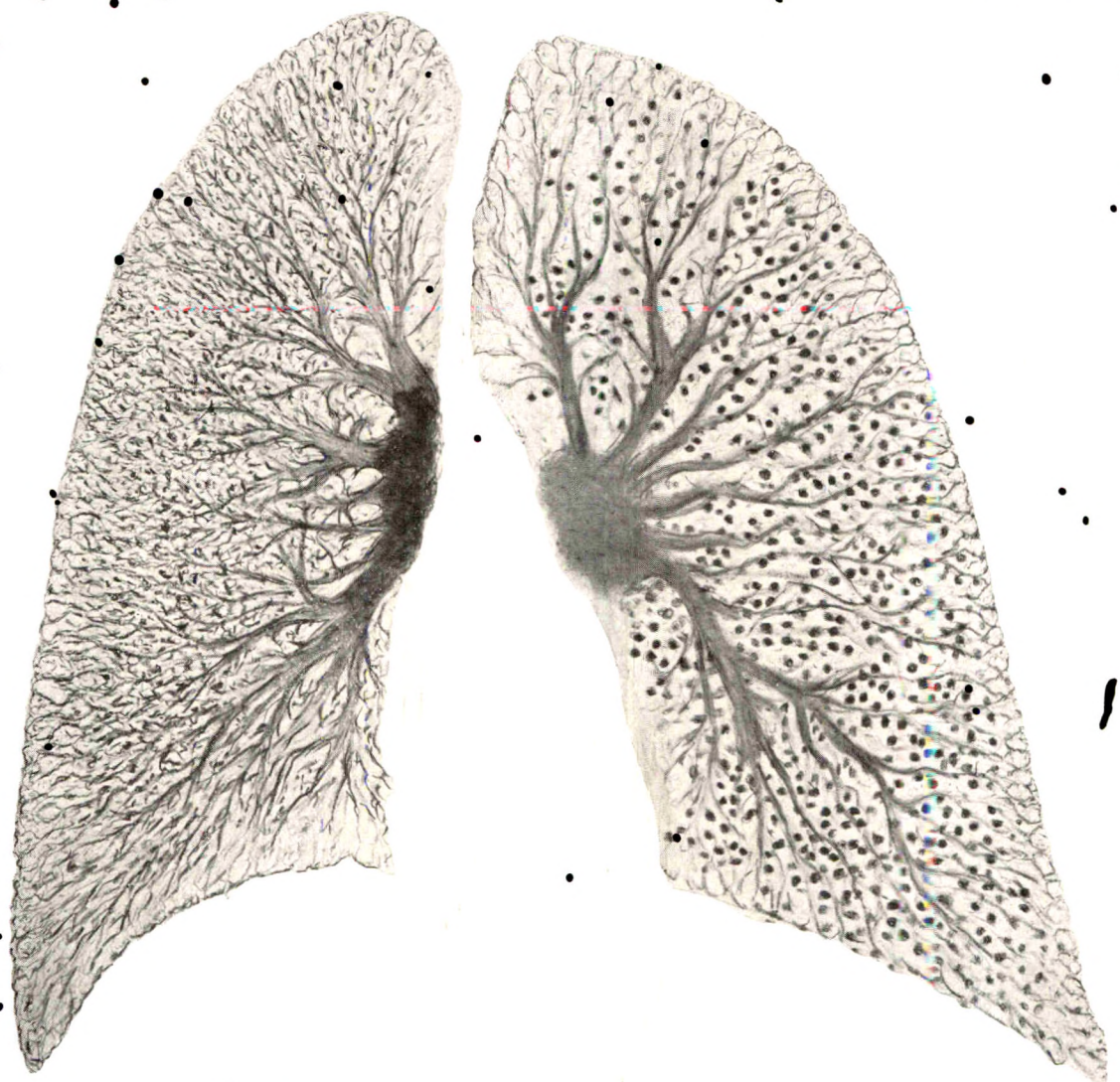


Fig. 22

Fig. 23

Fig. 22. *Vesicular type of pneumoconiosis* is evidenced by mottling with small, irregular shaped shadows that bear a definite relationship to the smaller blood vessels and bronchi, and tend to join one another. These shadows are cast by deposits of dust, dust laden phagocytes and collagen laid down as sheaths around the *smaller* blood vessels and bronchi, and in the stroma of the lung between the terminal lung lobules. These findings are not new; they were termed "M.F.U." (more fibrosis than usual) by Dr. Smith of South Africa, and as "fine nodulation" or "ground glass appearance" by others, but often they are not differentiated from the nodular type of silicosis.

Fig. 23. *Nodular silicosis* is evidenced by small, round, clear cut shadows that bear no relationship to the blood vessels and bronchi as they radiate from the hilum, and they are separated from one another by relatively well ventilated lung; these findings are readily differentiated from those observed in the vesicular type, as illustrated in Figure 22. These shadows are cast by collagen laid down in whorls, like the layers of an onion. This is the conventional type of lesion on which the diagnosis, and even the definition, of silicosis has been based.

1. Nodules or whorls, with clear cut, well defined edges; these whorls of collagen remind one of a cross section of an onion.

2. Laminae. Collagen may be laid down in laminae, as sheaths surrounding the blood vessels and bronchi.

3. Spheroidal masses or "gobs" of collagen, in irregular shapes, are laid down

largely in the deep layers of the pleura.

4. Strands, that is, fine strands of collagen, laid down in the walls of the alveoli or in the stroma surrounding the primary lung lobules.

None of these patterns of collagen is similar to any of the patterns of connective tissue. The deposition of the collagen is, I believe, a protective mechanism. Collagen is present in connective tissue but it acts as a filler between the cells. It reminds one of wax poured into a container filled with eggs; the yolks of the eggs correspond to the nuclei and the whites of the eggs to the cell bodies; the blue wax fills the chinks between the cells.

Relatively young connective tissue cells have very large nuclei and relatively clear cell bodies (Fig. 19). The spaces between these cells are filled with the blue stained collagen, which acts as a filler.

As this young connective tissue becomes older, the cells become fusiform and eventually are very greatly elongated, and the nuclei diminish in size. The waxy collagen filler between these cells is altered in shape to correspond with the aging characteristics of this connective tissue. None of the patterns of the aging reparative connective tissue is like any of the four previously mentioned patterns of collagen, as observed in pneumoconiosis or silicosis.

This finding is of more than academic interest because chronic inflammatory and tuberculous lesions of long standing develop connective tissue which contracts and deforms the thoracic cage and retracts the trachea to the side affected, whereas massive accumulations of collagen, no matter how old, do not do this. The only place in the lungs of silicotic patients in which connective tissue grows is within the lumen of the blood vessel, and this subject will be discussed later.

The normal gross and microscopic anatomy of the lung is essential, and is considered in our complete text, but space does not permit of its consideration here. A tracing of the pulmonary artery (Fig. 20) must serve as a substitute for this.

SIX TYPES OF PNEUMOCONIOSIS

To correlate the morbid changes in the lung that are manifest by dust deposits, masses of collagen, cell proliferation, air cysts, and intravascular deposits with the roentgen shadows caused by the wide va-

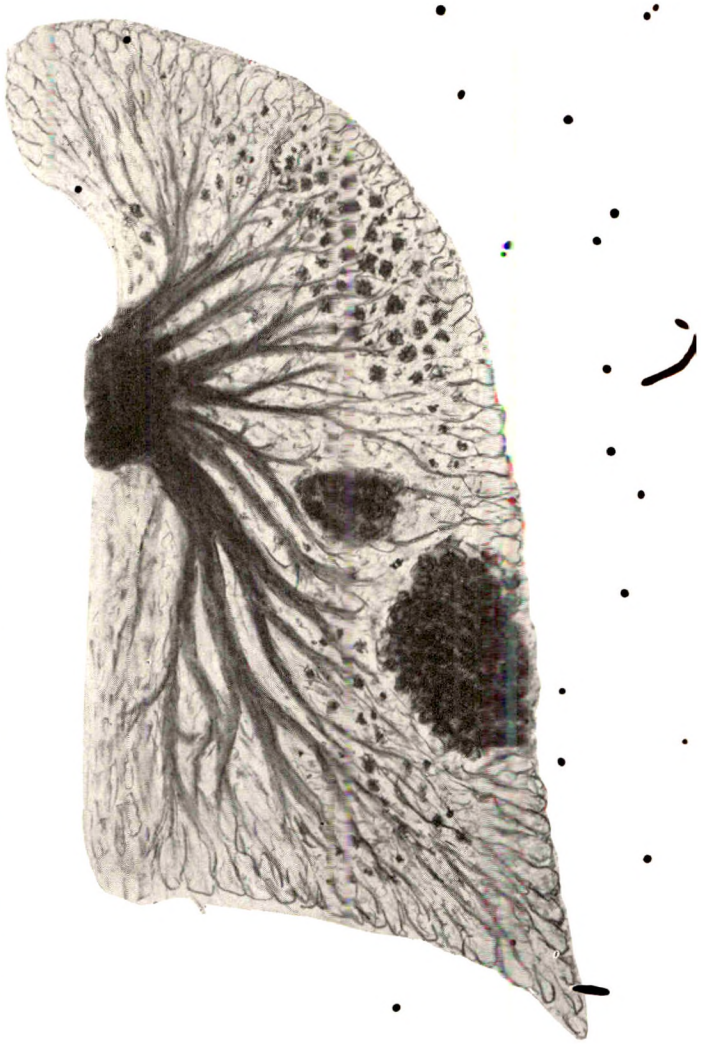


Fig. 24. *Tuberculoid pneumoconiosis* is evidenced by a mottling that is so similar to that caused by pulmonary lesions of the acid-fast bacillus origin, that the two cannot be differentiated from each other roentgenologically or by microscopic examination of hematoxylin and eosin stained sections. Even the distribution of the dust lesion tends to coincide with that of the acid-fast bacillus origin. However, it can be established that this lesion is caused by dust and not by the acid-fast bacillus of Koch.



FIG. 25. *Cystic type of pneumoconiosis* (previously described in our preliminary report as "pock marking type") is shown on the original roentgenograms by black spots on a white background. This is the direct antithesis of the nodular type, which is evidenced by white spots on a black background.* These black spots are air cysts that are caused by a dilatation of the terminal lung lobules that have become dilated because of the ball-valve action of deposits of collagen or dust, which encroaches upon the bronchioles and terminal air passages and allows the air to enter, but not to exit. The walls surrounding these cysts are thickened in contradistinction to the emphysematous blebs of asthmatic origin or compensatory dilatation.

riety of findings previously illustrated, six schematic drawings based partly on roentgenologic findings and partly on pathologic findings, are presented.

I. Peribronchial perivascular lymph node pneumoconiosis of Pancoast (Fig. 21).

II. Vesicular pneumoconiosis involving the terminal blood vessels and bronchi (Fig. 22).

III. Nodular pneumoconiosis, popularized by the South African workers (Fig. 23).

IV. Tuberculoid pneumoconiosis (Fig. 24).

V. Cystic pneumoconiosis, referred to in our previous communications as "pock marking type" (Fig. 25).

VI. Parenchymal pneumoconiosis (Fig. 26), hitherto considered under the term "acute silicosis," "rapidly developing silicosis."

Each of the illustrations of these types has a complete explanation in the legend and therefore they will not be described in the text.

While these various types tend to overlap, we believe that social and economic justice will best serve employe and employer alike if they are considered as separate entities. In the manuscript of the book from which these data are extracted, these six types have been considered in great detail. The following descriptions, with a few illustrations, are only abstracts and of necessity cannot contain all of the data in support of our conception of this problem of dusty lungs.

1. *Peribronchial Perivascular Pneumoconiosis*

Terminology. Peribronchial perivascular lymph node manifestation is a term that was applied by Pancoast to a lesion of the lung caused by the inhalation of dust. This lesion is manifest roentgenologically by accentuation of the hilar and linear markings. Pancoast's broad approach to

* This drawing is reproduced as a positive and therefore the black spots on the original roentgenogram appear as white spots for more convenient comparison with the other five types.

the subject of dusty lungs is indicated by the title of his first paper, namely, "The effect of the inhalation of dust on the lungs." The question of whether this type of lesion should be considered as "silicosis" caused a cyclonic controversy resulting in the compilation of a definition tending to exclude it.

Etiology and Pathogenesis. In this type of lesion the inhaled dust that lodges within the alveoli is engulfed by phagocytes and transported through the lymph channels that run parallel with the blood vessels and bronchi, either toward the hilum or toward the pleura. Some of these dust laden phagocytes become blocked in the lymph channels at the hilum and others are dammed back into the lymph channels that run parallel with the larger and medium sized bronchi. As these phagocytes are lodged there they cause deposits of collagen (not connective tissue) to be laid down in laminae as sheaths around the larger and medium sized blood vessels and bronchi, as if they were wrapt in crinkled cellophane.

A schematic drawing (Fig. 24) based partly on roentgenologic findings and partly on pathologic findings illustrate my conception of peribronchial perivascular pneumoconiosis.

Roentgen Findings. The morbid changes just described as they are outlined against the air in the adjacent alveoli appear as accentuated hilar and linear markings (Fig. 27; 28 and 3). The increased density of the lung caused by these morbid changes increases the opacity of the mid lung field. This lesion is usually bilateral, relatively symmetrical, and is often accompanied by increased opacity at both bases and perhaps at both apices. This variation in density is best observed by squinting the eyes so that one does not see the structures and simply notes the degree of blackness of the roentgenogram. This can be accurately recorded by a photometer, as has been previously described. The increased hilar shadows may be circumscribed or they may radiate out along the blood vessels and bronchi, like the tentacles of an octopus. At



Fig. 26. *Parenchymal pneumoconiosis* (hitherto defined as "acute silicosis" or "rapidly developing silicosis") is evidenced roentgenologically by a general haze or diffuse cloudiness which obscures the normal markings of the lungs. This general haze or diffuse cloudiness observed in the roentgenograms is caused by incomplete filling or incomplete consolidation of the alveoli and air passages with dust, phagocytes and hypertrophied cells. Collagen deposits in any pattern are extremely scanty in this type of lesion, even in its terminal stage.

Socially and economically these subjects present the most serious problem since roentgenograms of this type show no nodulation, therefore the tendency is to consider such cases as not silicosis, and therefore not within the law.

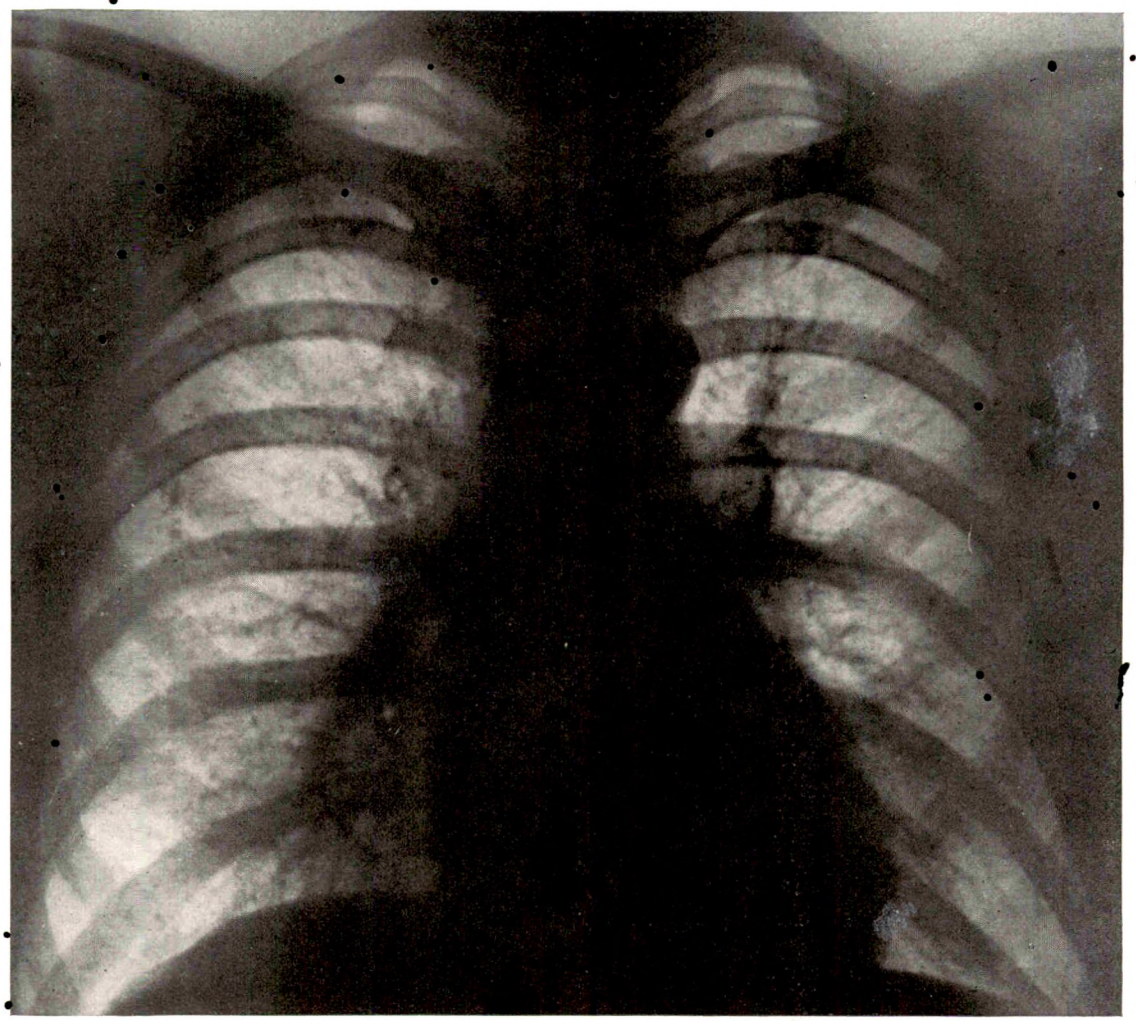


FIG. 27. (Lent by Dr. MacArthur of Noranda, Quebec.) *Peribronchial perivascular lymph node manifestation* (described by Pancoast) is evidenced roentgenologically by accentuations of the hilar and linear markings. The hilar markings may be circumscribed, or they may project out along the branches of the blood vessels and bronchi, like the tentacles of an octopus, as far as the middle and peripheral thirds of the lung. Dr. Smith of South Africa classified this particular roentgenogram as "more fibrosis than usual" and said that this lesion was found in 30 per cent of the men not working underground. A better example of this type of lesion is shown in Figure 3. These are definite manifestations of pneumoconiosis but often they are of little significance in the moderately advanced stages.

points where these increased linear markings overlap, their shadows on the roentgenogram are increased, giving a mottled appearance.

Gross Pathology. The mass at the hilum radiating out along the bronchi and blood vessels may be very dense and on gross section of the lung it may be so filled with grit that it is almost impossible to cut it with a sharp autopsy knife. These pathologic findings are best observed in large microsections that extend all the way from the hilum to the periphery of the lung, especially when they are stained with Masson's trichrome stain and used as a lantern slide.

The increased density at the hilum and around the bronchi is not due to connective tissue, and certainly not to reparative connective tissue with aging characteristics. When these structures are viewed mi-

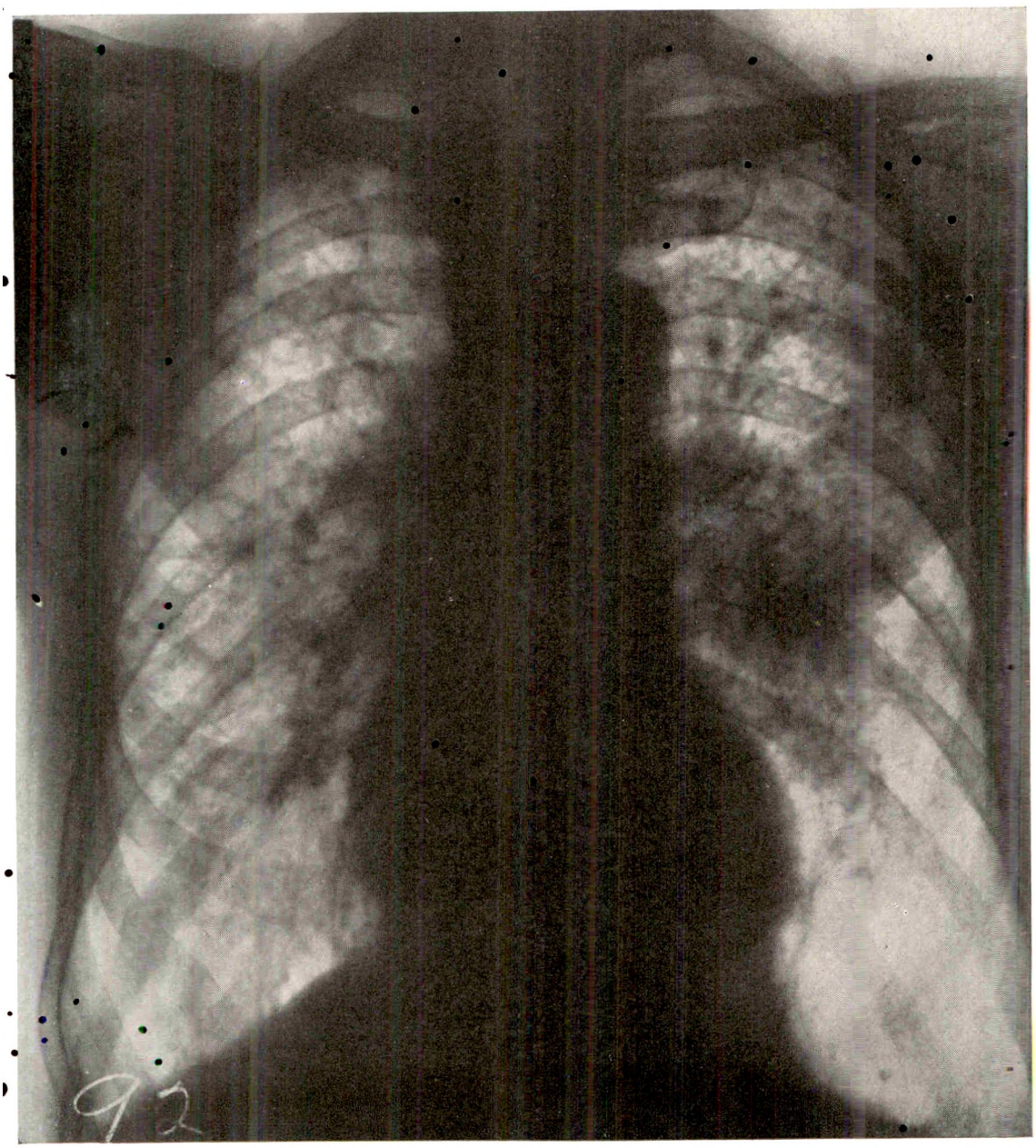


Fig. 28. In the late stages of the peribronchial perivascular lymph node manifestations, after quantities of dust have been phagocyted out of the alveolus and deposited at the hilum, around the *larger* blood vessels and bronchi, definite roentgen shadows are cast as shown in the right lung. Even this hilum, which is less involved than the left, was almost impossible to cut through with a sharp autopsy knife when sectioned for microscopic study. Socially and economically this class of lesion becomes a problem, first, because it constitutes such a large percentage of pneumoconiotic cases, and second, because it is a menace both to labor and to industry. The man who is perfectly able to work and wants to work may not be able to get a job because he has such a lesion. On the other hand, industry may be saddled with the compensation of a man with such a lesion, who is perfectly able to work, but does not want to work.

Grit at the hilum, when (cont. in Fig. 29)

croscopically they are observed to be composed of dust, both opaque non-refractive and transparent birefractive, dust laden phagocytes, collagen laid down in whorls or strands, and moderate cell proliferation. When the larger blood vessels and bronchi are cut transversely or obliquely, the sheath of dust laden phagocytes and collagen appears as a ring adjacent to the muscular coat (Fig. 30). The opaque flecks are observed in the conventional light field, and the transparent birefractive flecks of silica and the silicates, not seen in the light field, are observed when the field is rendered dark by cross polarization (Fig. 29).

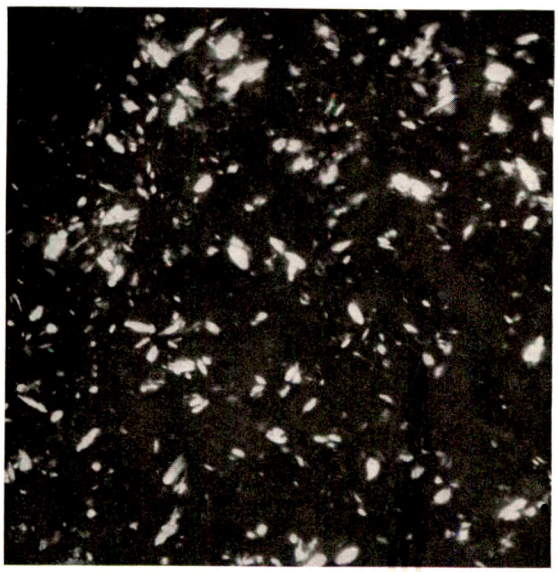


FIG. 29. Viewed with the field rendered dark by cross polarization, the huge quantities of silica crystals show why it was so dense and hard to cut.

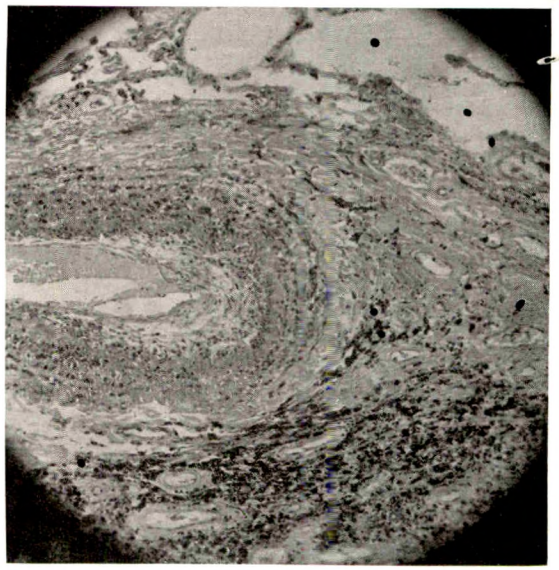


FIG. 30. Collagen above the edge of the blood vessel and dust laden phagocytes below form a sheath around an artery that is two or three times as thick as the arterial wall.

The vast majority of pneumoconiotic lesions are of this type, which is of less clinical significance than the other types of pneumoconiosis. Nonetheless, these are dust deposits and morbid changes incident to the inhalation of dust. This type of pneumoconiosis develops where there is a moderate amount of dust with a low percentage of silica or silicates. In our collection the most characteristic roentgenograms of this type occur in buffers and grinders, in miners in upper Maryland and lower Pennsylvania (loaned to us by Drs. Derr and Bridgers), and in the anthracite miners (loaned by Drs. Corcoran and Gordon of Scranton). Dr. Smith, of South Africa, whom the Canadian Government brought to their country to aid them in the solution of their silicotic problems, said that this type of lesion occurs in 30 per cent of the men not underground (but presumably inhaling some dust).

These cases rarely develop symptoms until the latter decades of life. An occasional case, with very slight roentgen findings and no dyspnea, will claim compensation. This procedure reacts unfavorably to his fellow workers, therefore these cases become potential social, economic and industrial problems. However, these problems must be faced and solved, if possible.

There is just as much injustice on the part of the employer or management who may refuse to employ a man because of

roentgen findings observed in pre-employment examination.

II. *Vesicular Pneumoconiosis*

Terminology. This term is not altogether satisfactory, but it is used as a suggestion, hoping that someone will offer a better one. Although this lesion is closely associated with the peribronchial perivascular lymph node manifestation just described, it is such an important factor in the problem of dusty lungs that we are loath to subjugate it to the term "peribronchial perivascular lymph node manifestation" which is under a cloud of disapproval since it was eliminated from the diagnostic, social and economic problems by the definition of silicosis hitherto discussed.

Etiology and Pathogenesis. The etiology and pathogenesis of the vesicular type of pneumoconiosis are identical with the previous type, except that the dust laden phagocytes and small strands of collagen are laid down in the lymph channels surrounding the *smaller* blood vessels, bronchioles and terminal lung lobules. These dust deposits around the terminal lung lobules, especially if they are black, are easily visible as a ring, many of which when joined together give a peculiar net-like appearance to the surface of the lung just beneath the pleura. This vesicular lesion is usually associated with the peribronchial perivascular type just described, but it may predominate to such an extent that it detracts attention from the involvement of the larger and medium sized bronchi and blood vessels. My conception of this lesion is illustrated in the schematic drawing (Fig. 22) previously presented in comparison with the other five types.

Roentgen Findings. The roentgen findings of the vesicular type of pneumoconiosis are evidenced by a bilateral, relatively symmetrical, diffuse mottling with a pattern not so clear cut and well defined as a silicotic nodule. These small roentgen shadows are irregular in shape, often like a Y or sections of a jig-saw puzzle. Prongs of one such shadow seem continuous with those of another. These small irregular shadows bear a definite relationship to the shadows cast by the smaller blood vessels and bronchi, which is the direct antithesis to the silicotic nodules which are separated from each other and *do not* bear a definite relationship to the blood vessels and bronchi. These roentgen findings are by no means new; they have been described by roentgenologists but frequently have not been differentiated from the nodular type of pneumoconiosis.

Roentgenologists have used such terms as "fine nodulation," "ground glass appearance," "M.F.U." (more fibrosis than usual) and "anteprimary," etc., to designate them. Smith of South Africa designated these as "M.F.U. + ++, +++." In their more advanced stages he designated them as "anteprimary +, ++, +++." These terms he applied to distinguish this lesion from the nodular type of silicosis to which the term "primary silicosis" was applied. These roentgen findings have usually been considered as early stages of nodular silicosis, but we believe that these lesions do not develop into typical nodular type of silicosis described in Type III. We believe that this lesion should be considered as a pathologic entity, perhaps associated with the mid lung peribronchial perivascular lesion, of which, as a matter of fact, it forms a part.

The gross appearance of these lungs differs very little from the normal and certainly very little from the peribronchial perivascular lymph node manifestations. The morbid changes are best illustrated in large microsections stained with Masson's trichrome stain projected onto the screen as a lantern slide.

In the mid lung field of such a section one observes a moderate sized artery and bronchus cut transversely (Fig. 32). These structures are surrounded by a sheath of dust laden phagocytes and laminae of collagen which appear as rings similar to that described and illustrated in Type I but they are smaller. A small branch of this medium sized artery cut longitudinally gives the key

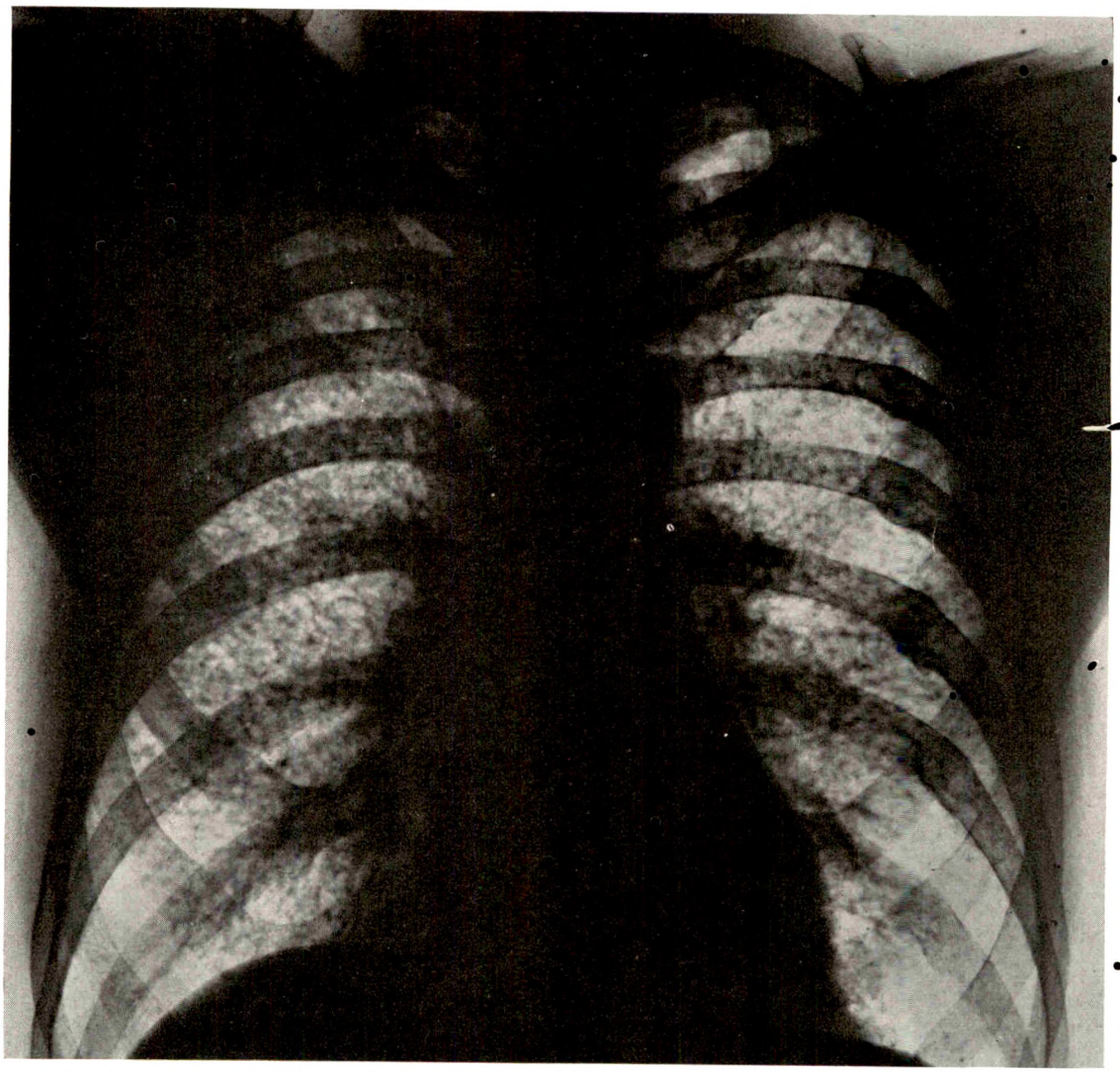


FIG. 31. (Lent by Dr. Gordon, Scranton, Pa.) *Vesicular Type of Pneumoconiosis. Roentgen Findings.* Bilateral, symmetrical mottling of small, irregular spots that are joined together but do not have clear cut and well defined edges. These irregular spots bear a definite relationship to the terminal blood vessels and bronchi. These findings are not new; Dr. Smith of South Africa referred to them as "M.F.U.+, ++, and +++," in their earlier stages, and as "anteprimary silicosis+, ++, +++," in their more advanced stages, thus distinguishing them from the nodular, which he considered "primary silicosis." These small, irregular spots, characteristic of the vesicular type of pneumoconiosis are caused by (cont. in Fig. 32)

for correlating the vesicular type of pneumoconiosis with the mid lung lesion described by Pancoast and illustrated in Figures 3 and 27.

This terminal blood vessel cut longitudinally shows the artery for its entire length (Fig. 32). The dust sheath which surrounds it appears as parallel black lines that lie adjacent to the outer surface of the muscular coat. This black sheath corresponds almost identically with the characteristic mottling observed roentgenologically; even the bulging is similar to the beading observed in the roentgenograms. The fact that these thick walled blood vessels and bronchi cast shadows on the roentgenogram that are so definitely associated with the pattern of the normal lung differentiates

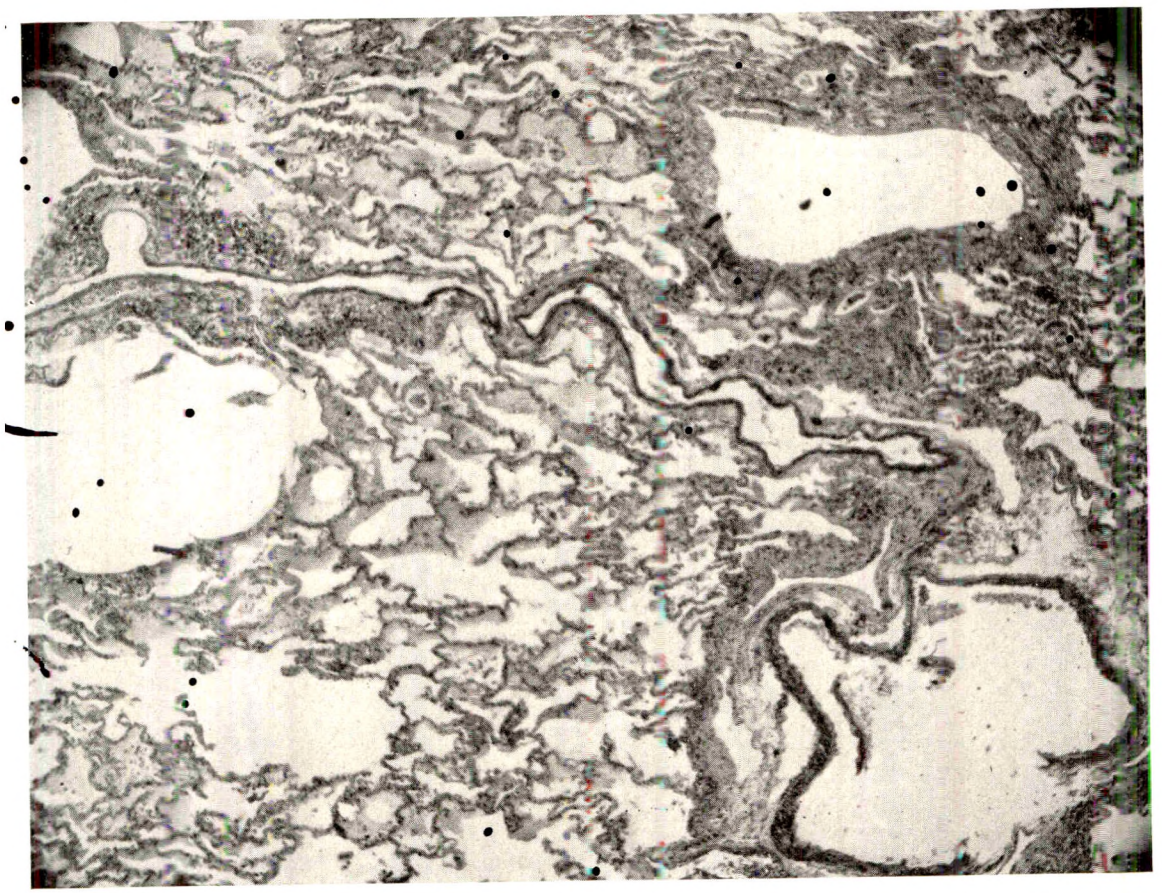


Fig. 32. Deposits of dust laden phagocytes and collagen laid down in sheaths around the smaller blood vessels and bronchi; even the bulging or budding at the left of the illustration is similar to the roentgen findings. Dilated lung lobules are seen to the left, and a medium sized artery and bronchus cut transversely, with sheaths of collagen and dust, are seen to the right.

them from the nodular whorls (silicotic nodules) which are denser, rounder and bear no relationship to the blood vessels and bronchi. Compare Figures 22 and 23 of the schematic drawings and Figures 31 and 33 of the roentgenograms for the similarity and differentiation of these two types of lesions.

The terminal lung lobules supplied by these partially constricted or contracted or compressed bronchioles or air passages tend to become distended (Fig. 32) and in this respect they somewhat overlap the type of pneumoconiosis to which we have applied the term 'cystic." These cases will therefore be discussed in greater detail under that heading. I believe that this type of lesion is of greater clinical, social and economic significance than the peribronchial perivascular type involving the larger and medium sized bronchi. I have seen one case of this type with extreme dyspnea, far out of proportion to the density of the lung. The alveoli were markedly dilated. There was clubbing of the fingers with deformity of the nails due to interference with the pulmonary circulation.

III. *Nodular Pneumoconiosis (Silicosis)*

Terminology. The term "nodular silicosis" has become so firmly fixed in the medical literature that it may seem audacious even to limit its application to the specific lesion it was originally intended to describe. This term was given the stamp of approval by the Committee which drafted the defini-

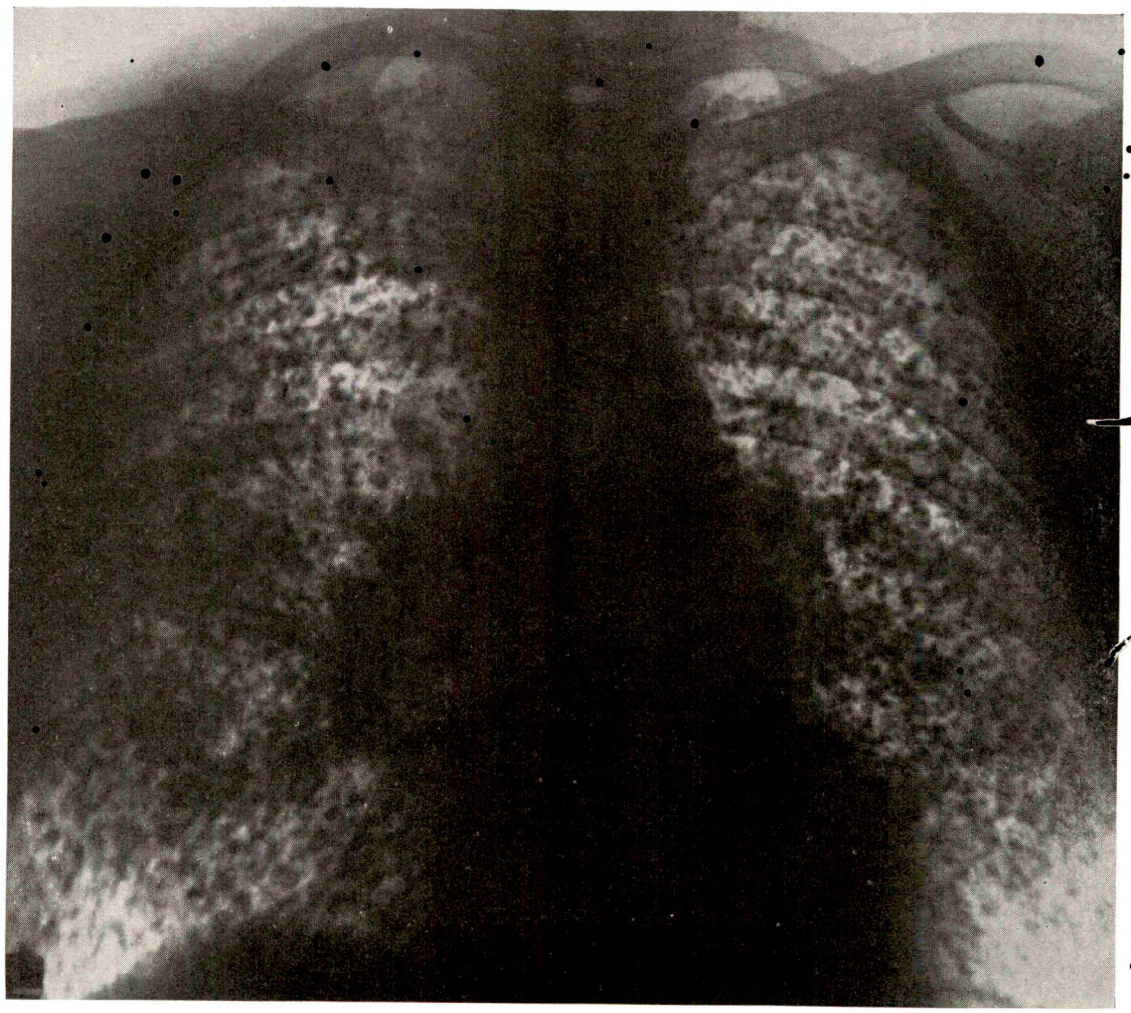


FIG. 33. (Lent by Dr. Klein, Perth Amboy, N. J.) *Nodular Pneumoconiosis. Roentgen Findings.* Bilateral, symmetrical mottling, with small round spots having clear cut edges, that bear no relationship to the radiating blood vessels and bronchi; in the earlier stage they are separated from one another by well ventilated lung. There they may coalesce, especially in the right lower mid lung field where the three lobes overlap, producing the "pawnbroker's sign."

Clinical Manifestations. Although these lungs are shot full of nodules, the patient was free from symptoms; the lesion was found accidentally while he was being examined for fractured ribs. Absence of symptoms in this type of lesion is the rule rather than the exception.

Socially and economically many of these subjects are able to work at hard labor. Should they be prevented from getting a job or losing the one they have? Legislative acts are primarily built around this type of lesion hitherto considered silicosis, in contradistinction to the two previous types. The small, round, clear cut spots, characteristic of this type of lesion, are caused by (cont. in Fig. 34)

tion of silicosis published in the *U.S. Public Health Bulletin,* in August, 1935. No better term could have been found for these small, isolated, spherical masses in the lung, and therefore in this communication we are limiting the term "nodular" to the specific lesion it was specifically intended to describe. This type of lesion was originally described, or at least popularized at the turn of the century, by the South African workers. Whether they intended to limit this term to nodular whorls (or silicotic nodules), which they considered to be due to fibrous nodules of connective tissue, is not

clear. But the definition published in the *Public Health Bulletin* is fairly specific, in limiting silicosis to this type of lesion. There is some doubt in my mind, if those who formulated the definition differentiated the nodular type of silicosis from the vesicular type, on the one hand, or the chronic tuberculoid type, on the other.

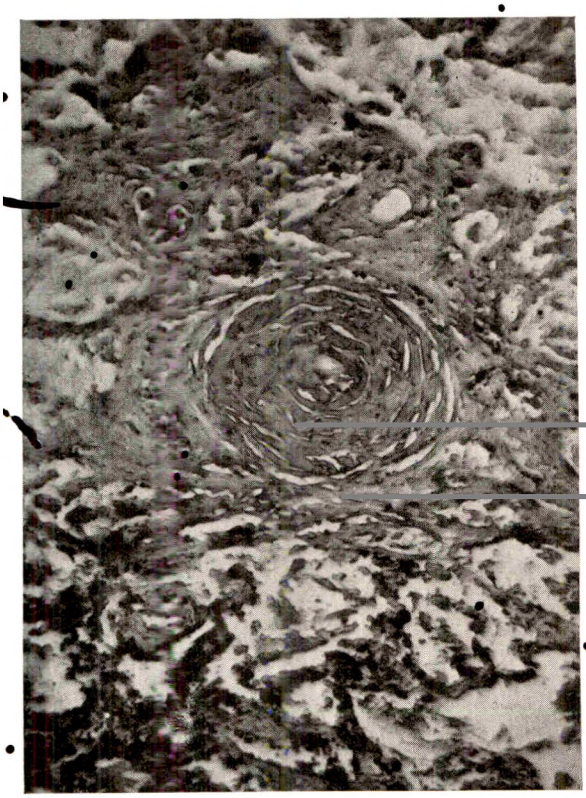


Fig. 34. Whorls of collagen laid down like the layers of an onion. These small spherical masses which are clear cut in outline have been termed "silicotic fibrous nodules," but they are not composed of connective tissue.



Fig. 35. These nodules are supposed to develop in lymph follicles, but such follicles are not only *not* the home of the nodule, but they are freer of collagen than any other structure of the lung. When whorls do develop near such a follicle it pushes the follicle aside.

Etiology and Pathogenesis. The etiology and pathogenesis of the nodular whorls to which the term "silicotic nodule" has hitherto been applied are not as clear as the etiological and pathological factors of the other five types. These nodules are not connective tissue nodules; certainly they are not composed of reparative connective tissue with aging characteristics of the cell. They are composed of collagen laid down like the layers of an onion (Fig. 34), and adult nodules contain very little dust, either opaque nonrefractive or transparent birefractive flecks within the interstices of these layers of collagen. In what we believe to be the nascent nodule, that is, the young early developing nodule, there are more deposits of dust and dust laden phagocytes and less collagen than in the adult nodule. This evidence seems to indicate that the nodular whorl of collagen (so-called silicotic nodule) constitutes a protective mechanism in which clumps of dust laden phagocytes are incarcerated within the interstices of these layers of collagen.

If this theory is true, the question of what happens to the dust within the phagocyte as the whorl becomes of age is pro-

lematic. It has hitherto been stated and rather universally accepted that lymph follicles throughout the lung are the location of the silicotic nodules or collagen whorls. Our evidence definitely tends to prove that these lymph follicles are not only *not* the home of the nodule, but that nodules are less likely to occur in the lymphatic follicles than in any other histologic structure of the whole lung. Indeed, when a nodular whorl develops adjacent to a follicle, it pushes the follicle out of the way instead of growing into it, indicating that the two are incompatible (Fig. 35).

A schematic drawing compared with the others helps to convey my conception of this type and to differentiate it from the other types.

Roentgen Findings. The term "nodulation" or "roentgen-ray nodulation" has hitherto been rather loosely applied to shadows cast by pathologic structures other than the silicotic nodule or collagen whorls. If we were to speak of bilateral symmetrical mottling rather than nodulation, and if one looks for the points of differentiation between the different types of mottling, it becomes evident that there is a difference between the clear cut, well defined round or oval shadows that are bilateral, relatively symmetrical and bear no relationship to the blood vessels and bronchi (Fig. 33) and the irregular shaped mottling described under the term "vesicular pneumoconiosis" (Fig. 31), and that both of these should be differentiated from the pock marking type, characterized by black spots on a white background, as observed in the original roentgenogram. These black spots on a white background are negative shadows that are cast by accumulations of air in the cystic type of pneumoconiosis and will be considered under the cystic type.

Microscopic Examination. Viewed microscopically two or more whorls of collagen may be connected to form conglomerate nodules, and groups of nodules may be connected by strands of collagen to form small localized areas of dense lung. Viewed with higher power, the nodule is observed to be composed of layers of collagen that are relatively free of nuclei (Fig. 34). It has clear cut, well defined edges and is relatively free of dust, either opaque or transparent birefractive flecks. When the nodules are viewed with the microscope field rendered dark by cross polarization, they are often singularly free of silica, compared with the surrounding structures.

Social and Economic Problems. This type of lesion is slow in developing, perhaps because the nodules form a protective mechanism. The patients are often singularly free of symptoms, even when the lungs are filled with these nodular whorls of collagen (Fig. 33) (silicotic nodules).

IV. *Chronic Tuberculoid Pneumoconiosis*

Terminology. Tuberculoid pneumoconiosis is a lesion that looks like pulmonary tuberculosis but is not caused by the acid-fast bacilli of Koch. Certain other terms herein used are defined as follows:

Dust Nodule: This term is applied to small, irregularly spherical masses that are relatively free of collagen, but are composed of opaque, non-refractive and transparent, birefractive flecks of dust, that usually have a definite relationship to the peripheral blood vessels and bronchi.

Silicotic Nodule: The term "silicotic nodule" is applied to whorls of collagen widely disseminated throughout the lungs, bilateral, relatively symmetrical, that bear no constant relationship to the blood vessels and bronchi.

Dust Tubercle: This term, in contradistinction to the dust nodule on the one hand, and the silicotic nodule, on the other, is applied to irregularly spherical masses composed of collagen combined with dust laden phagocytes that is so similar in appearance to the pulmonary tubercle of acid-fast bacillus origin that the two can scarcely be differentiated by microscopic examination of hematoxylin and eosin stained sections, but special stained sections show that the dust tubercle contains no acid-fast organisms.

Acid-Fast Pulmonary Tubercle: This

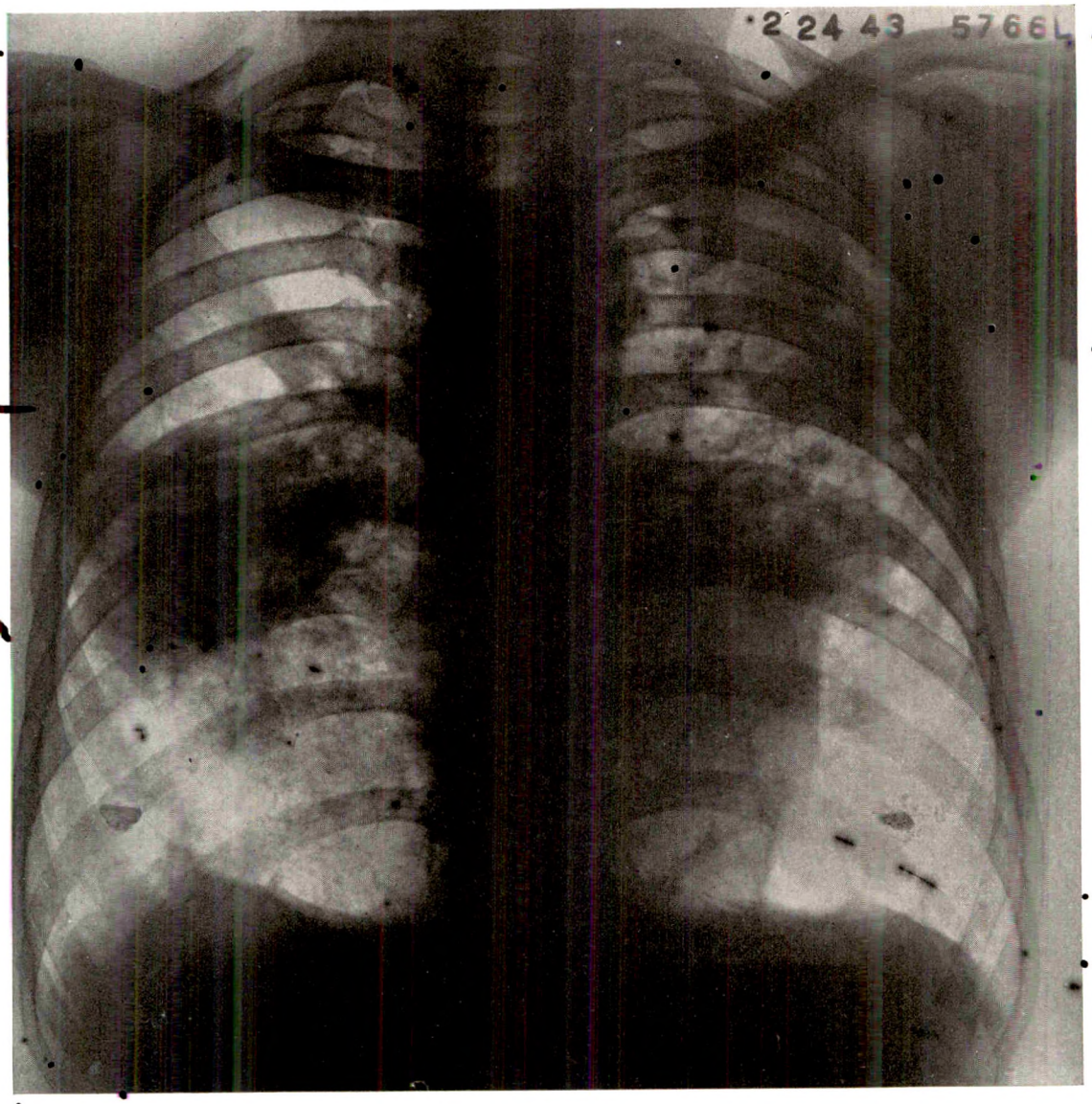


Fig. 36

term is reserved for pulmonary tubercles that can be proved to be caused by the acid-fast bacilli of Koch.

This tuberculoid lesion has not hitherto been differentiated from nodular silicosis, on the one hand, and silicotuberculosis, on the other.

Etiology and Pathogenesis. Reverting to the anatomy and histology of the lung and to the travelogue of the phagocyte hitherto recorded, it becomes evident that the phagocytes emerge from the alveolar wall and engulf foreign body flecks. These phagocytes may not all progress through the lymphatic channels toward the hilum or toward the pleura. In the tuberculoid type of pneumoconiosis these dust laden phagocytes congregate in an alveolus or in adjacent alveoli and together with collagen form small, irregularly spherical masses that correspond in size, shape and structure to the pulmonary tubercles of acid-fast bacillus origin.

A schematic drawing of this type (Fig.

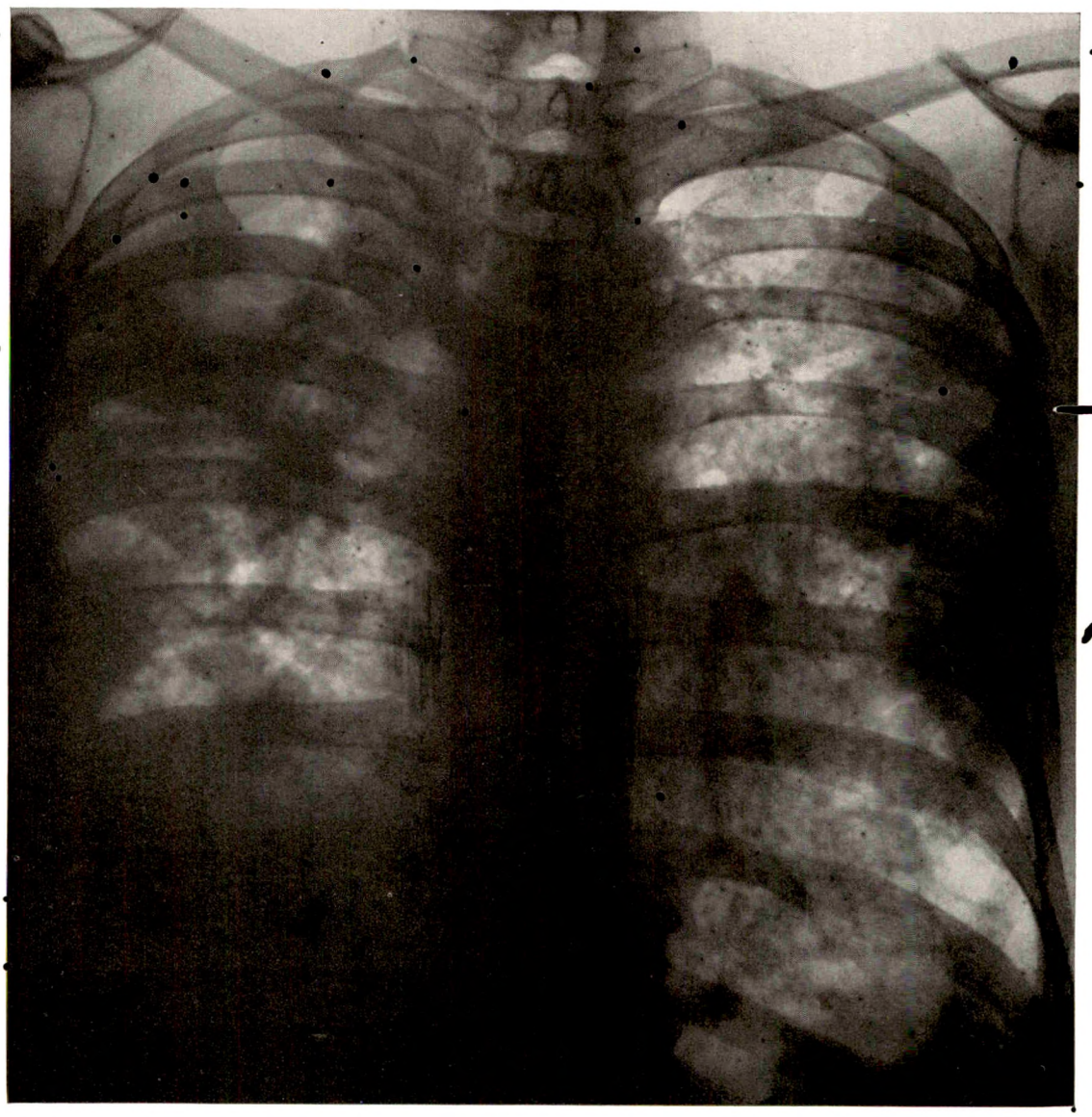


Fig. 37

24) aids one in differentiating it from the other five types, but it does not aid in its differentiation from pulmonary tuberculosis of acid-fast bacillus origin.

Roentgen Findings. The outstanding characteristic of the roentgen findings (Fig. 36) is that they so closely simulate findings observed in pulmonary tuberculosis of acid-fast origin that the experienced roentgenologist, specialist in tuberculosis, and even experts in the problem of pneumoconiosis and silicotuberculosis, would unhesitatingly state that the patient was suffering from pulmonary tuberculosis. They might qualify this statement by saying that if there were an authentic history of adequate exposure to silica, the case might be considered as silicotuberculosis. This lesion is usually bilateral, is by no means symmetrical, and often there are large, dense areas in one or the other apex, with areas of infiltration of various sized nodules or tubercles in the mid lung field of the opposite lung. These tubercles or nodules bear no relation-

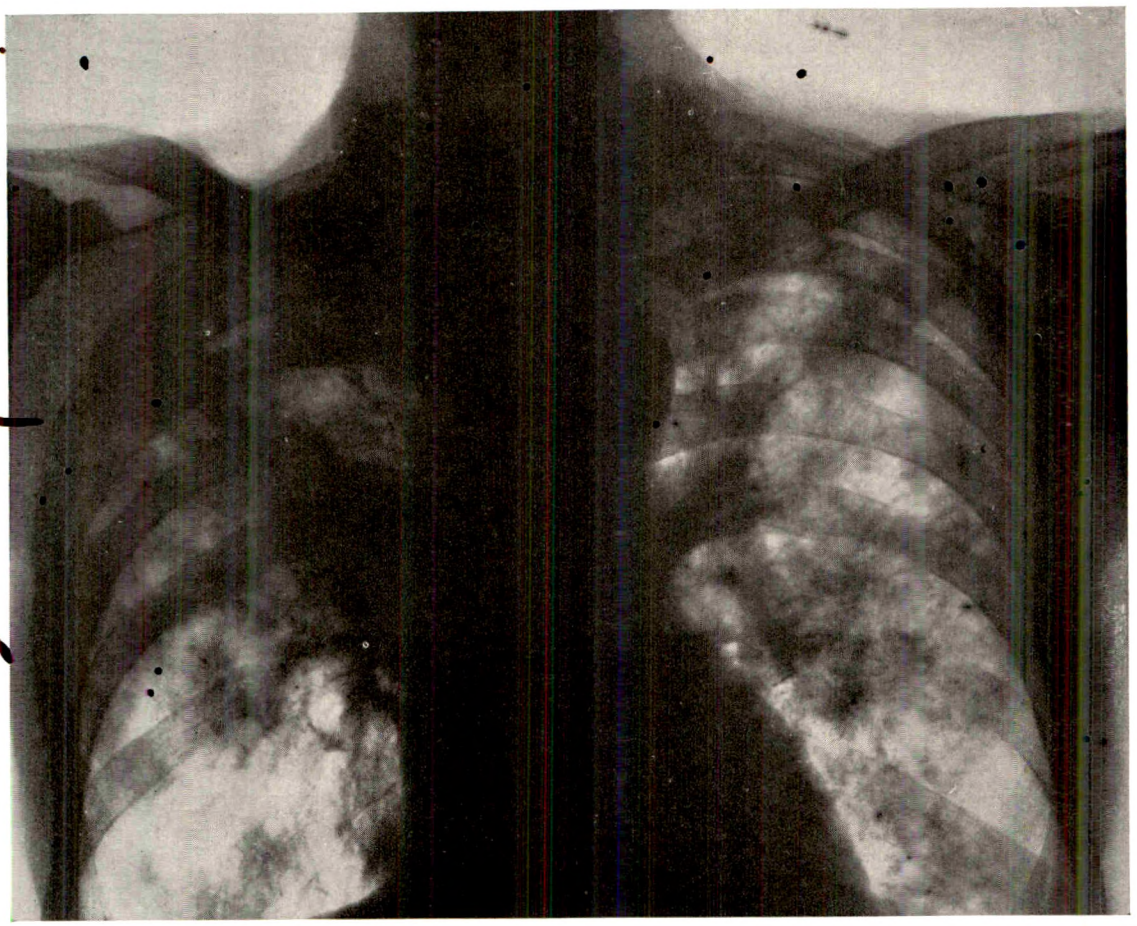


Fig. 38

Fig. 36,* 37†, 38‡. *Tuberculoid Pneumoconiosis.* This is a lesion which resembles pulmonary tuberculosis but is not caused by the acid-fast bacillus of Koch.

Figures 36, 37 and 38 are illustrations shown over this continuous caption. One of these is a lesion caused by dust without acid-fast bacilli of Koch; another is caused by the acid-fast bacilli of Koch, without dust; and the other occurred in a man with a thirty-two year history of intensive exposure to dust in a mine, but he now has acid-fast bacilli in his sputum. To emphasize the similarity of these lesions and the difficulty of differentiating them, they are reproduced for comparison with one another, without at this time prevealing which is which.

* Referred by Dr. H. J. Noering, Valatie, N. Y.
† Referred by Dr. Harold Gartner, Valhalla, N. Y.
‡ Lent by Dr. William Corcoran, Scranton, Pa.

ship to the normal radiating linear markings.

Points of Differentiation. The roentgen findings that tend to differentiate tuberculoid pneumoconiosis of dust origin from the pulmonary tuberculosis of acid-fast bacillus origin are as follows:

1. With the dust lesion, the consolidated areas are homogeneous, even in roentgenograms made with the Potter-Bucky diaphragm.

2. The lesion lies closer to the periphery of the lung.

3. The heart shadow is rendered indistinct along its left border.

4. The trachea is only slightly deviated or retracted, if any.

5. The thoracic cage is symmetrical,

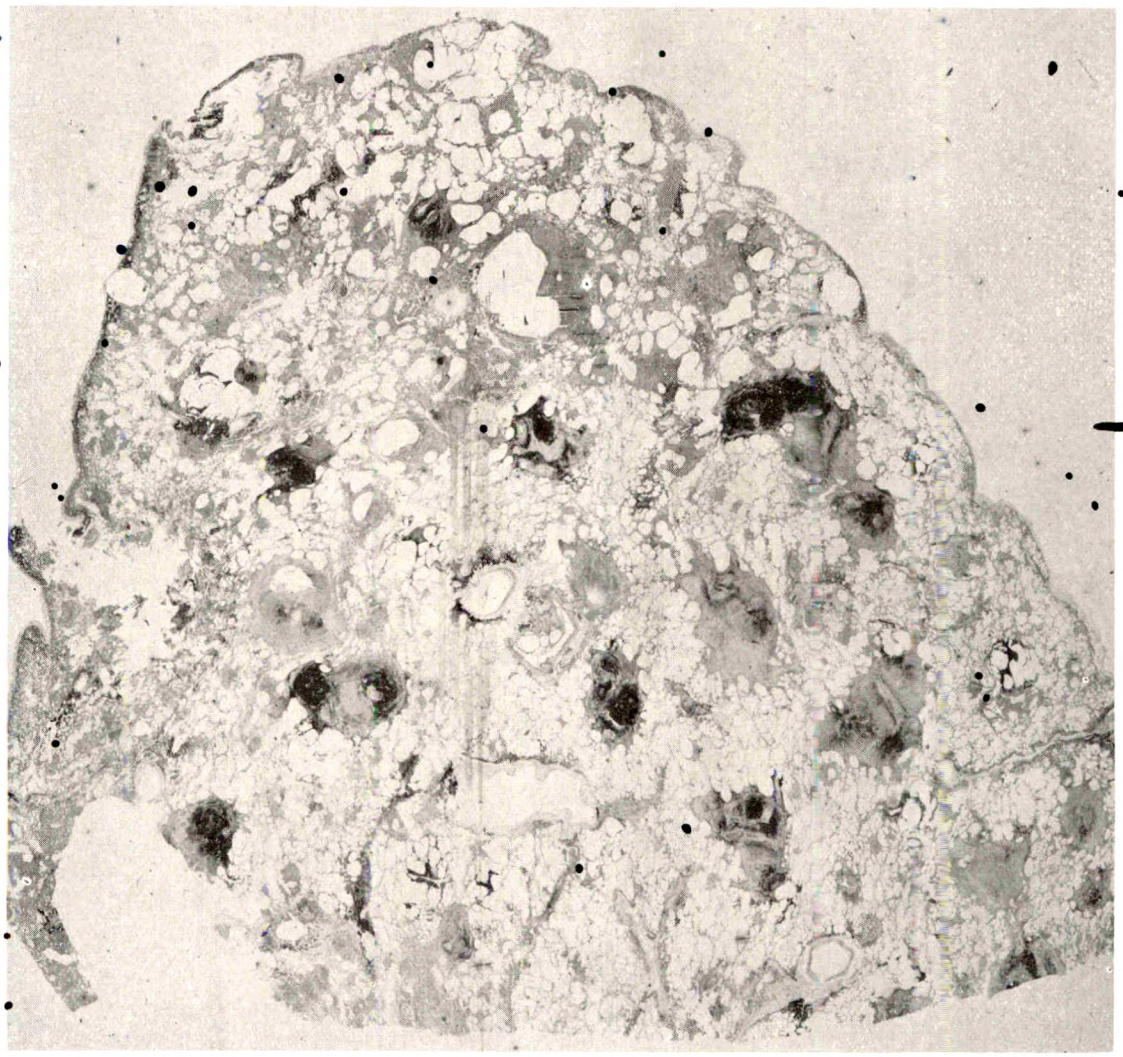


Fig. 39

large, and barrel shaped; the ribs are not drawn together, as is often the case in chronic fibroid tuberculosis.

A gross specimen, cut longitudinally, shows large and small dense areas widely disseminated throughout the lung.

Macroscopic examinations of large microscope sections, somewhat magnified and preferably thrown on the screen as a lantern slide, show the distribution of the tubercles of various sizes and shapes that are almost identical with the size, shape and distribution of tubercles of acid-fast bacillus origin. Two large microscope sections (Fig. 39 and 41), one of dust origin and the other of pulmonary tuberculosis of acid-fast bacillus origin, are included to show the great similarity between the tubercles of dust origin and those of acid-fast bacillus origin. Between these two illustrations we have inserted an illustration by Delafield (Fig. 40) in which he made an etching on stone showing pulmonary tubercles as observed with low power magnification. In our illustrations we have not designated which of the two (Fig. 39 or 41) is of acid-fast bacillus origin and which is of dust origin. For the time being we have left that to the reader in order to impress him with the similarity of the lesions.

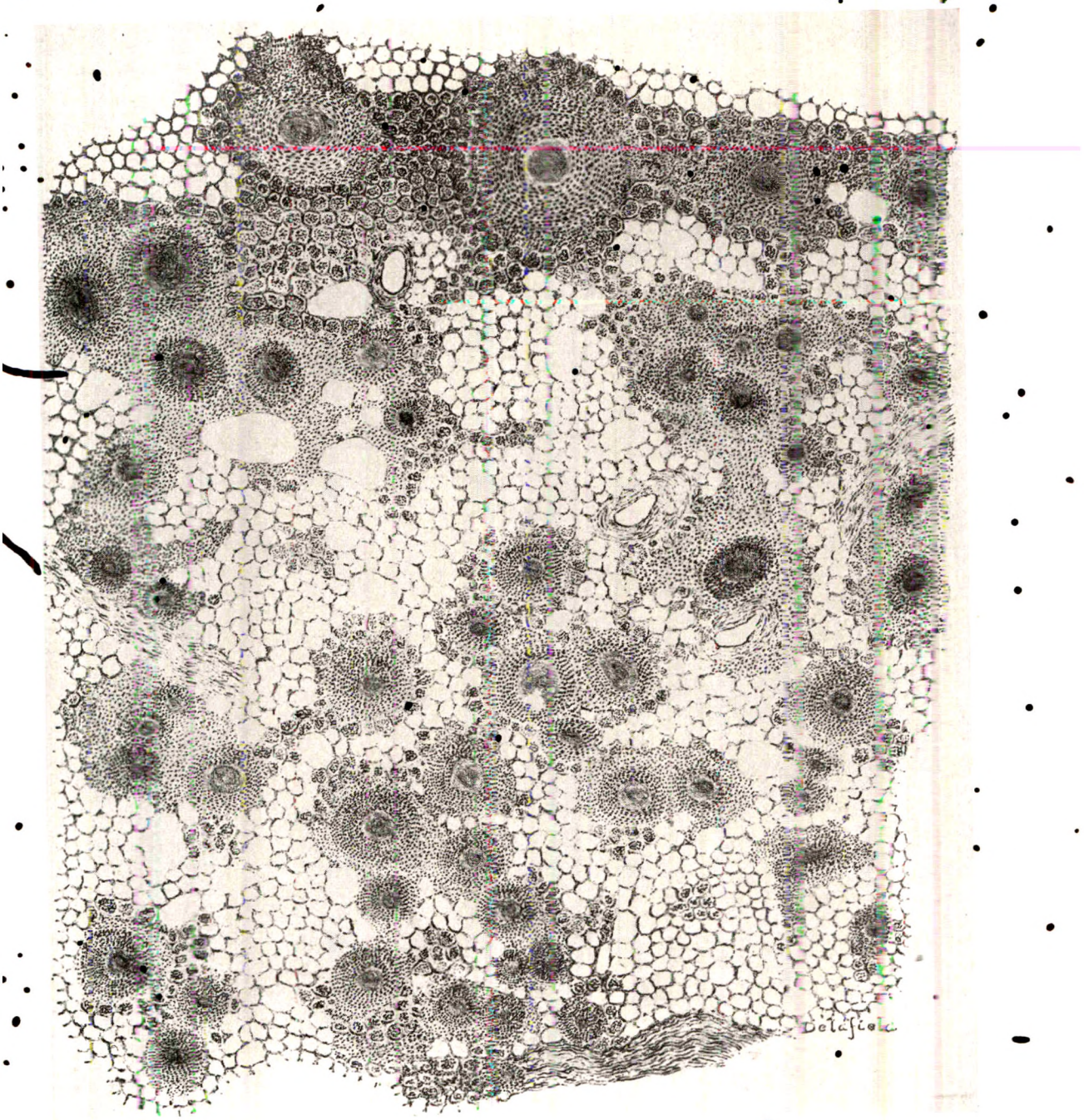


FIG. 40

Two medium power photomicrographs, one of an acid-fast bacillus tubercle and the other a dust tubercle, are also shown, but likewise, in proof of their similarity it is not revealed at the present time which is which.

Problem of Differentiation. A large microscope section, well stained with hematoxylin and eosin, was presented to a high ranking pathologist interested in this subject. He expressed the opinion that the lesion was silicotuberculosis. When it was suggested to him that it was silicosis and

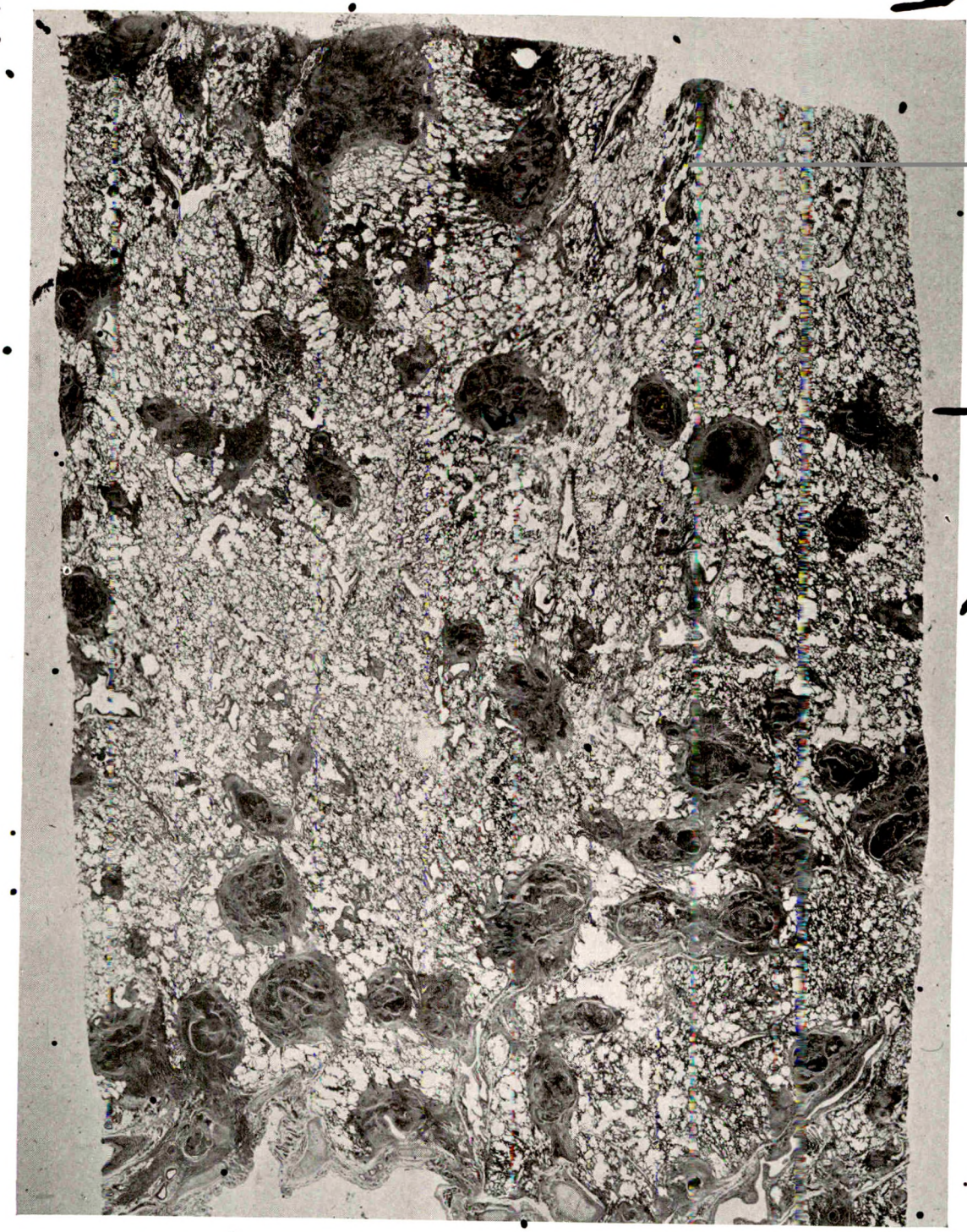


FIG. 41

FIG. 39, 40 and 41. Small irregular spherical masses, separated from one another by relatively normal lung. One of these is a reproduction of a drawing made by Dr. Francis Delafield which he personally etched on stone to illustrate the findings which he observed in pulmonary tuberculosis. This drawing of pulmonary tubercles was made before the discovery of the acid-fast bacillus was announced by Koch in 1882; one of the other two lesions is caused by dust without acid-fast bacilli, and the other by acid-fast bacilli. To emphasize the similarity of these dust and acid-fast bacillus tubercles, we are not now revealing which is which.

not silicotuberculosis, he reiterated his statement: "I mean silicotuberculosis." However, we believe that we have incontrovertible evidence obtained by special microscopic procedures and special stains to eliminate the possibility of this lesion having been caused by the acid-fast bacillus of Koch, and definite evidence that it is caused by dust, largely silica, of granite origin.

Three chapters in our book are devoted to the similarity between dust lesions and those of acid-fast bacillus origin, etiology and pathogenesis of both, and methods and criteria in proof of the fact that the tuberculoid lesion is of dust origin and not of acid-fast bacillus origin. In this article, it is impossible to present that evidence. We believe that this type of lesion is of more serious social, economic, industrial and legislative significance than are any of the previously described types.

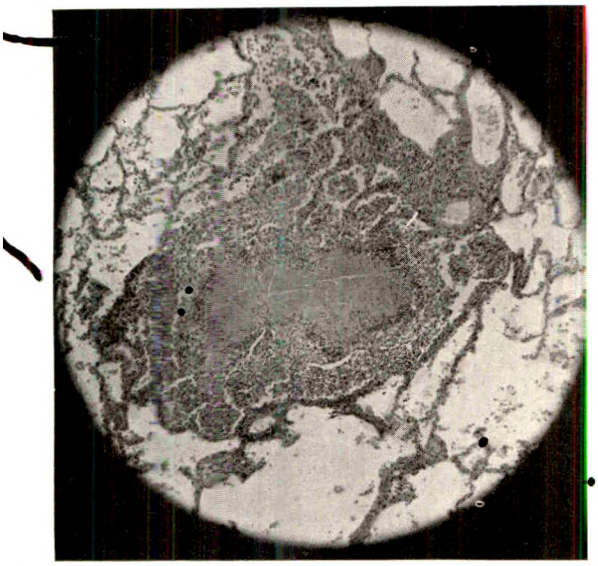


FIG. 42

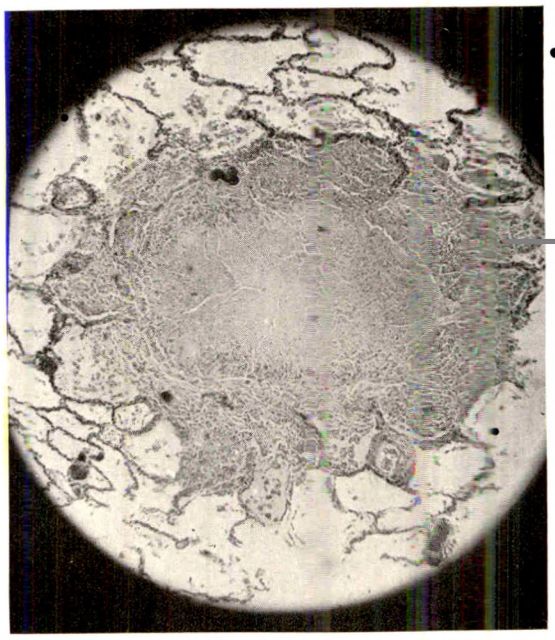


FIG. 43

FIG. 42 and 43. Even when these small, spherical masses, called tubercles, are studied microscopically with the conventional light field after being stained with hematoxylin and eosin, those that are caused by the acid-fast bacilli cannot be distinguished from those caused by silica of granite origin. More than sixty years ago Delafield described such types of pulmonary tubercles, but at that time the cause of pulmonary tubercles was unknown; the acid-fast bacillus of Koch had not yet been discovered.

The most classical of the seven types was the amorphous tubercle, the center of which was so disintegrated that its structures could not be recognized and therefore it was termed "amorphous." Surrounding this central part was a rim of viable cells. This type of tubercle is well illustrated in Figures 42 and 43, one of which is of dust origin and the other of acid-fast bacillus origin. Which is which?

v. *Cystic Pneumoconiosis*

Terminology. The term "pock marking" was applied to this lesion in our preliminary report on dusty lungs, but the term "cystic" is more scientific and more applicable. Cystic pneumoconiosis is evidenced by air cysts which are dilated pulmonary lung lobules. The bronchioles or air passages leading to the terminal lung lobule become occluded or compressed by masses of collagen or dust laden phagocytes which surround them or impinge upon them. This deposit of collagen and dust causes a valve-like action which allows air to enter the terminal lung lobule but does not allow its exit.

The schematic drawing (Fig. 25) is made in black and white to coincide with the other five types and even then it does not show the lesion as well as if it were pre-

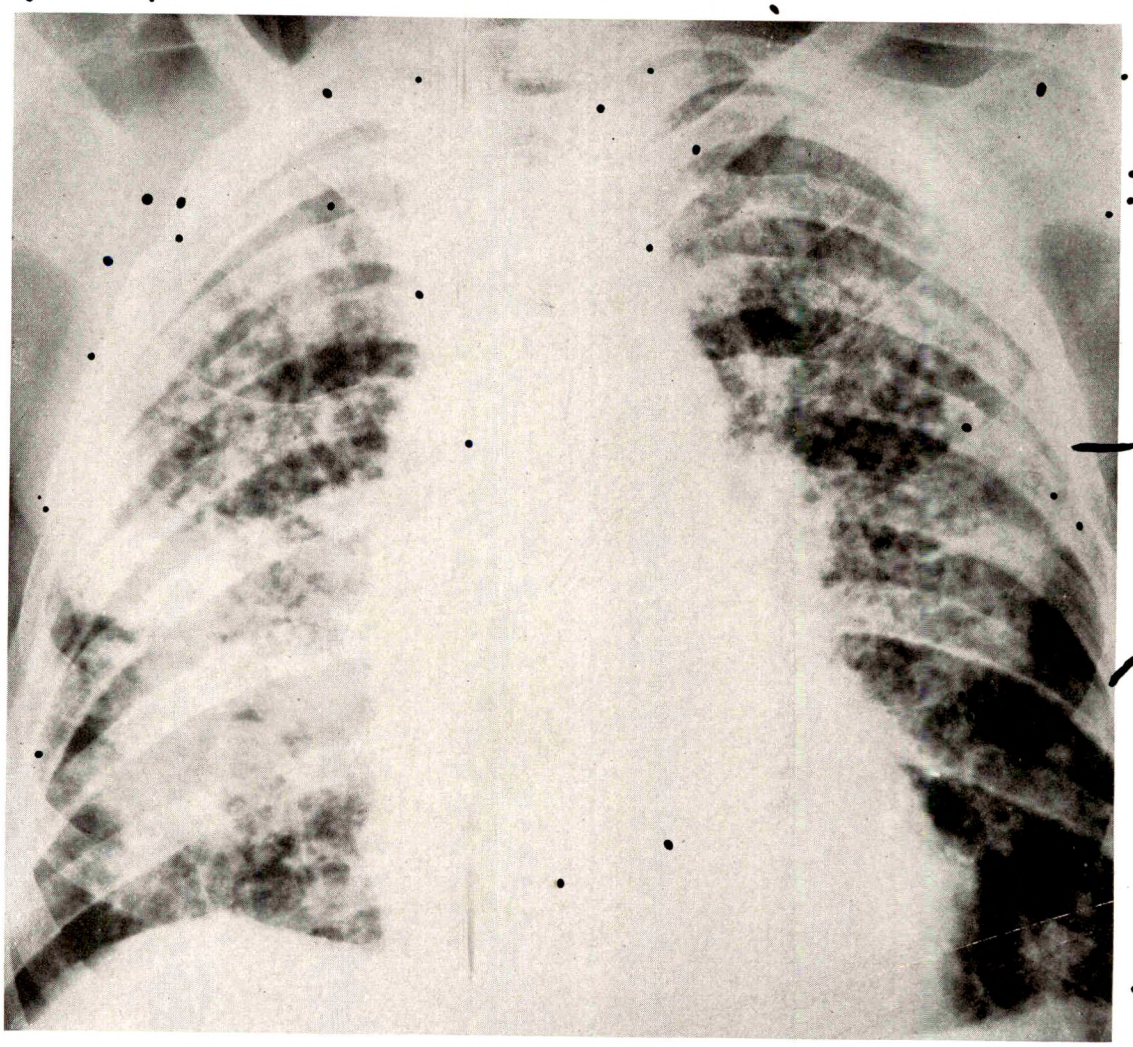


FIG. 44. *Cystic Pneumoconiosis* ("pock marking"). *Roentgen Findings.* Bilateral but not symmetrical mottling evidenced by small black spots on a white background, or surrounded by a white ring, is best shown in the second and third interspaces. This illustration is reproduced as a negative because the black spots on a white background are more readily seen than white spots on a black background. The black spots are air cysts caused by dilated terminal lung lobules; air is inhaled but its exit is blocked by the ball-valve action of collagen and dust deposits. Quantities of dust and collagen deposits are laid down in these lungs, some in the form of definite whorls, but in the original roentgenograms the black spots within white areas predominate. Socially and economically these constitute a much more serious problem than any of the previous types, except possibly the late stages of tuberculoid type.

sented as a negative drawing. This is done because it is much easier to see a black object on a white background, and for this reason all other roentgenograms are reproduced as positives rather than negatives, because it is the black or dense objects which constitute the roentgen findings in which we are interested.

Roentgen Findings. These spherical cysts that contain air within relatively solidified portions of the lung are surrounded by rings of dense tissue, cast black spots on a white background (Fig. 44), the direct antithesis of the roentgen appearance of the nodular type of pneumoconiosis (Fig. 33), which is manifest by white spots on a black background in the original roentgenograms. These lesions are bilateral, often are not as

symmetrical as the first three types, and while they do contain white spots caused by nodular whorls of collagen or deposits of dust, yet the cystic appearance predominates.

Gross Specimen. On the cut section of the gross specimen, these cysts appear as black spots located largely in its peripheral third of the lung.

Magnified photomicrographs of large microscope sections show these air cysts as round black spots in a white area (Fig. 45) that remind one of a slice of Swiss cheese; the black spots correspond to the holes in the cheese. A comparison of a negative print of a large microscope section (Fig. 45) with the roentgenogram (Fig. 44) emphasizes the great similarity of the pathologic and the roentgenologic findings.

When air cysts are observed in less dense regions of the lung, they may be surrounded by rings of collagen and these morphologic rings cast ring-like shadows in the roentgenograms. When these air cysts are viewed microscopically with low power magnification, the partition forming the alveolar wall within the lung lobule is compressed and as the air cyst dilates it, it likewise compresses adjacent alveoli that are displaced peripherally (Fig. 46). These air cysts are not to be confused with emphysematous blebs, although the mechanism that causes it is similar to the *modus operandi* of the emphysema of asthma except that in asthma the lesion is functional whereas in the cystic pneumoconiosis it is organic. Likewise, the dyspnea of asthma is intermittent or periodic, whereas the dyspnea of this cystic type is constant.

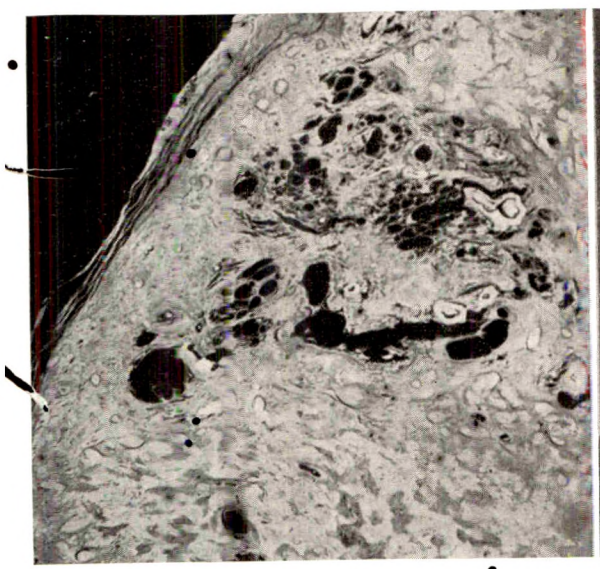


Fig. 45. A negative print of a microscopic section which is comparable with the roentgen findings; that is it shows black spots on a white background. It reminds one of a slice of Swiss cheese. Large and small cysts are seen, one of which is shown microscopically in Figure 46.



Fig. 46. This shows how the air compresses the partition within the terminal lung lobules. Adjacent alveoli surrounding the air cysts are compressed and deformed. These are to be differentiated from emphysematous blebs.

Social and Economic Problems. This type of lesion constitutes a more serious social and economic problem than any of the previous types, except possibly the advanced stages of the tuberculoid type.

Less distinct cysts that are caused by terminal lung lobules that are not definitely dilated but are somewhat surrounded by strands of collagen, as described in the vesicular type, are somewhat similar to these air cysts, but the terminal lobule is not so

much dilated and the rings surrounding it are less distinct than in the cystic type. Such terminal lung lobules filled with air but without thick walls are observed in Figure 32 which illustrates the dust sheath around the terminal blood vessels and bronchioles. This manifestation of the lesion is recognized in many roentgenograms if one looks for it. However, dust, dust laden phagocytes and collagen are not the only morbid changes which may cause these roentgen findings, and therefore air cysts themselves are not pathognomonic of pneumoconiosis, but they are very suggestive.

VI. *Parenchymal Pneumoconiosis (acute silicosis, so-called acute silicosis, or rapidly developing silicosis)*

(A) Acute Pneumonic Parenchymal Pneumoconiosis and (B) Acute Tuberculoid Pneumoconiosis.

Terminology. Parenchymal pneumoconiosis has hitherto been termed "acute silicosis," "so-called acute silicosis," or "rapidly developing silicosis." This type of pneumoconiosis (silicosis) was first described by MacDonald, Piggot and Glider, and then by Chapman of the Massachusetts General Hospital, with the roentgenograms lent to me by Dr. George W. Holmes. This lesion was later discussed by Gardner under the term "so-called acute silicosis." It seems wiser to us to discuss this lesion under the two headings of this section, viz., acute pneumonic parenchymal pneumoconiosis and acute tuberculoid pneumoconiosis, as being comparable with the acute pneumonic type of pulmonary tuberculosis or acute phthisis, in contradistinction to acute general miliary tuberculosis where the tubercles are small and separated from one another by relatively healthy lung tissue.

Definition. Acute pneumonic parenchymal pneumoconiosis is a lesion manifest by incomplete filling of the alveoli with dust, dust laden phagocytes, cholesterol, the clefts of which are observed in the microsection, and cells desquamated from thickened alveolar walls. Certain large areas in the lung are rendered almost completely avascular whereas adjacent portions of the lung are hypervascular; at least the capillaries are immensely dilated, containing many times the normal content of blood. This type of lesion is singularly free from nodular whorls of collagen (silicotic nodules) and there is relatively little collagen of any pattern in any portion of the section except in the walls of the alveoli where there are fine strands that have a tendency to occlude the pulmonary capillary circulation.

A schematic drawing (Fig. 26), fully described, is presented adjacent to the other five types to illustrate the characteristic roentgenologic and pathologic findings.

Roentgen Findings. The roentgen findings are manifest by a diffuse general haze or cloudiness, involving both lung fields (Fig. 47). This lesion is bilateral but usually not symmetrical. Within these areas where there is a general haze, there are localized patches of increased density that suggest more advanced degrees of consolidation. These lungs are singularly free of clear cut, well defined nodules on which the diagnosis of silicosis is usually based and unless one is familiar with this type of lesion he would scarcely even think of this being silicosis.

Such was the experience of those who encountered the Gauley Bridge group of cases, most of which were of this type.

Macroscopic Examination. The magnified microsections serve to correlate the roentgenologic and pathologic findings, and when a large section stained with Masson's trichrome stain is thrown on the screen, collagen is conspicuously absent (Fig. 48). A low power microscopic examination of large sections demonstrates the scarcity of collagen whorls (silicotic nodules) and a very slight amount of collagen that is present in any form. In the section stained with trichrome, the areas which are hypervascular stand out in brilliant red contrast to the avascular areas in which one can find scarcely a single red blood cell when the section is examined with higher power.

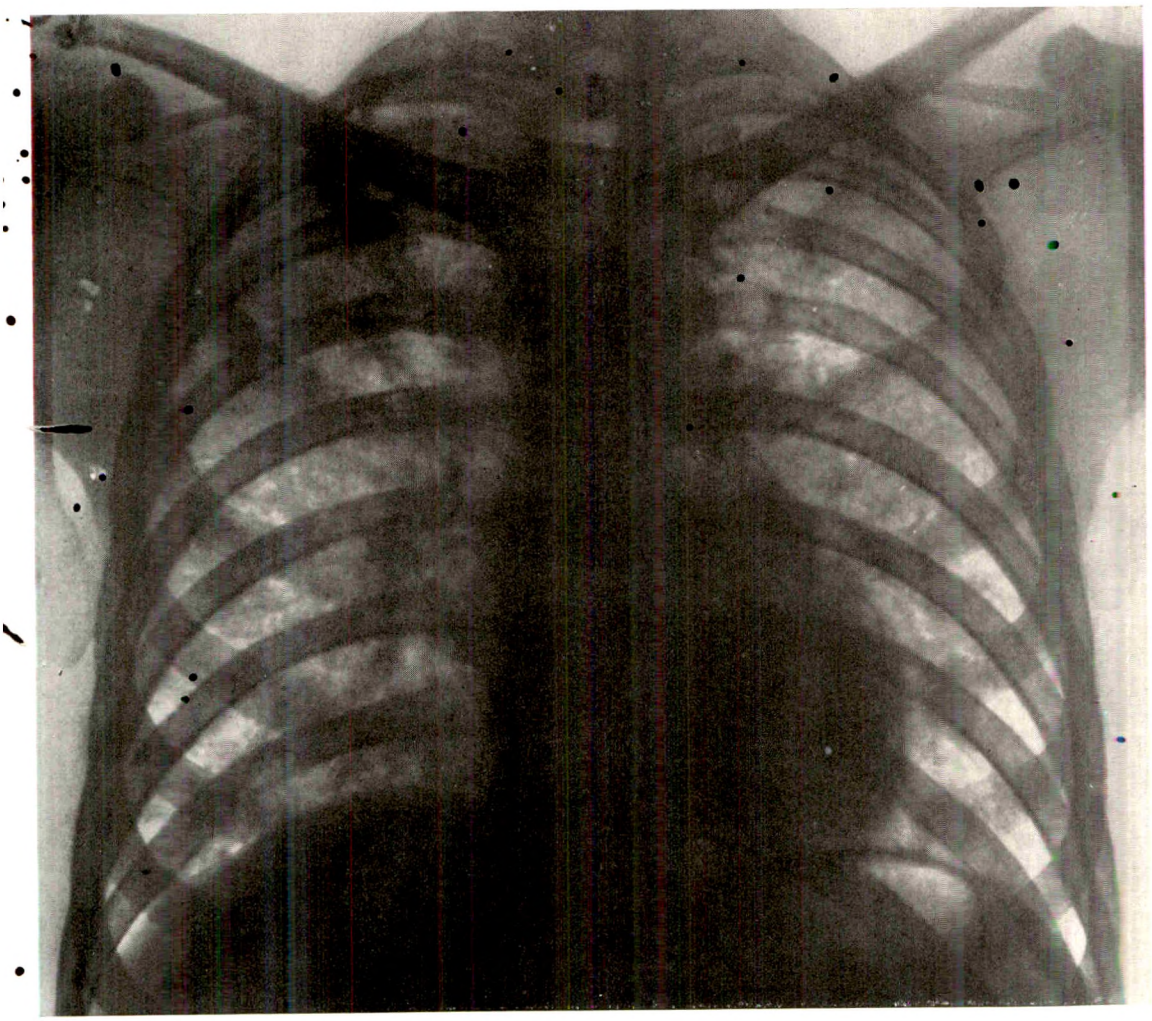


FIG. 47. (Lent by Dr. Harless, Gauley Bridge, W. Va.) *Parenchymal Pneumoconiosis* (acute silicosis or rapidly developing silicosis). *Roentgen Findings.* This is shown by a general haziness, without characteristic nodulation, and without accentuation of the linear markings. These findings are bilateral but not symmetrical. Owing to the absence of nodulation or accentuated linear markings, lesions of this type may not be recognized by the roentgenologist unless he is familiar with it.

Clinical and Occupational History. Male, aged about twenty-two; had twenty months' intensive exposure to dust with a high silica content. He was dead within twenty months from his first exposure.

Socially and economically, these subjects present the most serious problem and in the past injustice has been done to this group of men because the subjects were unfortunate in that they did not have the well developed nodulation on which the diagnosis and definition of the disease are based. The indefinite haziness in these cases is caused by (cont. in Fig. 48)

Microscopic Examination. The walls of the alveoli in the avascular areas are markedly thickened; the cells are hypertrophied and many are proliferated and desquamated. The alveoli contain quantities of dust, dust laden phagocytes composed of both the opaque nonrefractive and transparent birefractive dust flecks. These dust laden phagocytes are also observed in the stroma of the lungs. Fine strands of collagen laid down in the walls of the alveoli occlude the capillary circulation, and as we suggested in our first article on "The dyspnea of silicosis, what causes

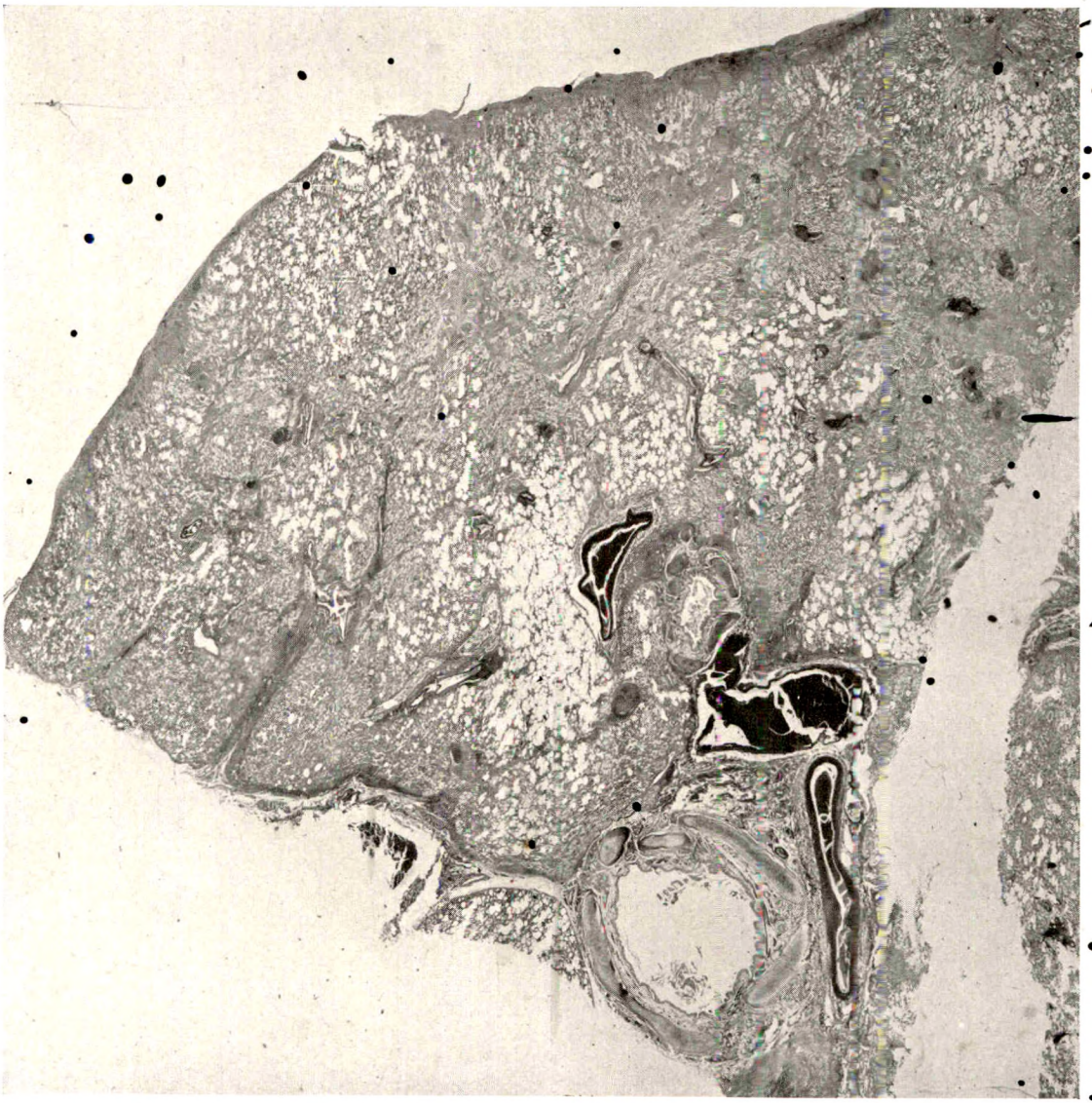


Fig. 48. Incomplete consolidation or filling of the alveoli and terminal air passages of the lung with dust, dust laden phagocytes and hypertrophied, desquamated cells from the walls of the alveoli. Collagen in any pattern is singularly scanty. This statement is hard to prove in this reproduction in black and white, but the absence of collagen in the original trichrome stained section is most convincing. Some portions of the microscopic section are avascular, whereas adjacent regions are hypervascular, as is elaborately described and illustrated in the full manuscript of our book, from which this is taken.

it?" we believe that this capillary occlusion is one of the causes, perhaps the principal one. In the hypervascular areas which lie adjacent to the avascular areas, the walls of the alveoli are thinned, the cells are not hypertrophied or proliferated, and these alveoli contain relatively little dust, either opaque or transparent, birefractive.

Acute tuberculoid pneumoconiosis is a lesion similar to that of the acute pneumonic manifestation (acute silicosis) in that the lesion is composed of dust, dust laden phagocytes and desquamated cells deposited in the alveoli and terminal air passages. Like the pneumonic lesion, collagen whorls (silicotic nodules) or collagen in any pattern are singularly scanty except



Fig. 49

perhaps as fine strands of collagen in the walls of the alveoli.

Acute tuberculoid pneumoconiosis is similar to the *chronic tuberculoid type* in that its distribution is the same, namely, the dust laden phagocytes congregate or aggregate in small, irregularly spherical masses that are similar in size, shape and distribution to those of acute miliary tubercles of acid-fast bacillus origin. Indeed, the lesion is so similar to that of acid-fast bacillus origin that we are using two low power photomicrographs (Fig. 49 and 50); one is a lesion of dust origin and the other of acid-fast bacillus origin, and to impress upon the reader the similarity of these lesions, we shall not at this time reveal which is which.

A similar field viewed with low magnification and by the conventional light field method shows a mass of phagocytes clumped together to form a tubercle (Fig. 51). No opaque dust is observed in these phagocytes, even by high power magnification, but when the field is rendered dark by cross polarization, thousands of transparent

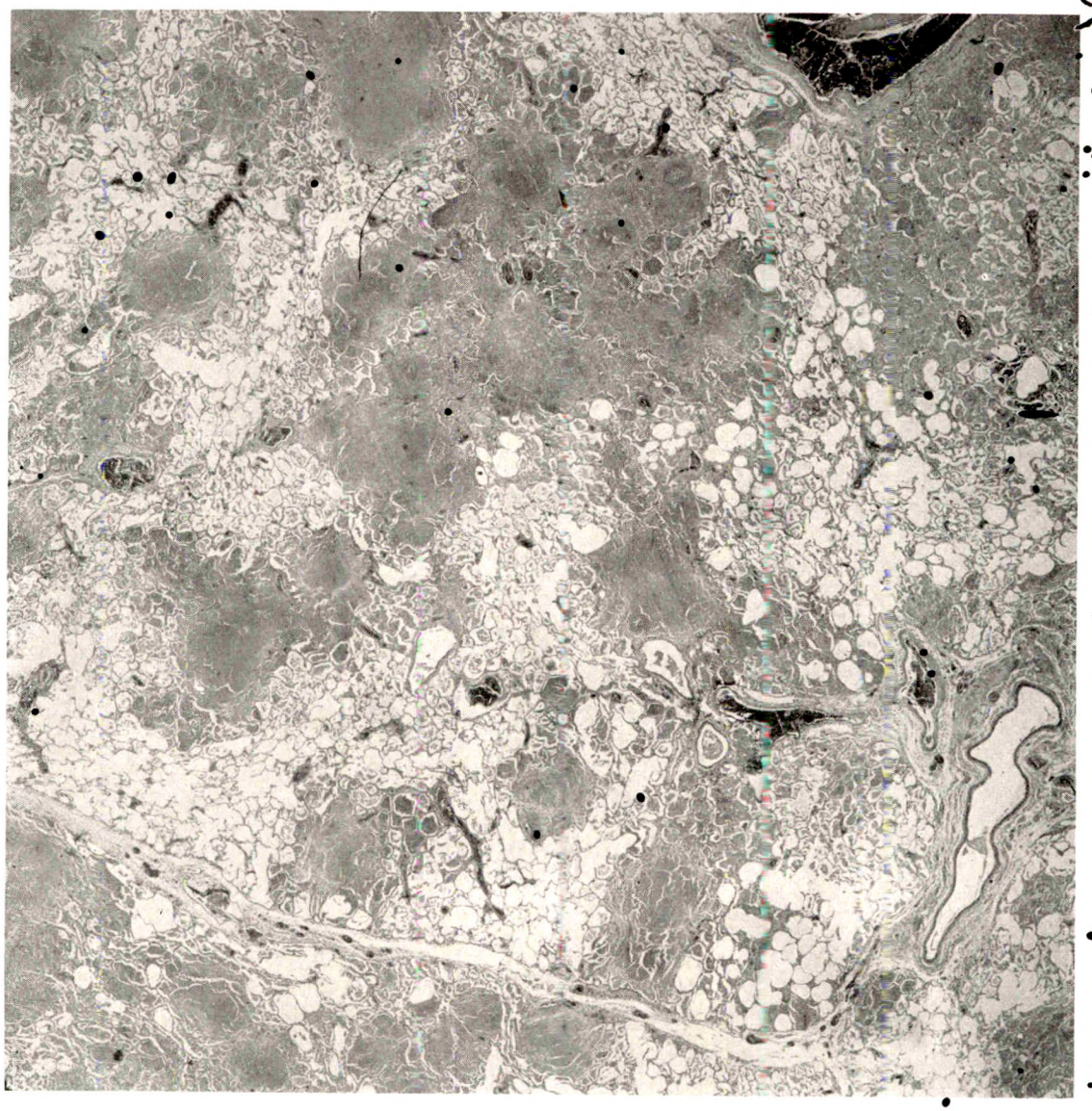


FIG. 50

FIG. 49 and 50. Acute pulmonary tuberculosis of acid-fast bacillus origin and acute pulmonary tubercles of dust origin are so similar that they cannot be differentiated from each other by microscopic examination of hematoxylin and eosin stained sections with the conventional light field method of examination. Microscopic examinations of these sections stained for the acid-fast bacilli showed that the pulmonary tubercles in one were literally loaded with acid-fast bacilli, whereas the pulmonary tubercle in the other case was singularly free of acid-fast bacilli. We are not yet stating which is which.

flecks become startlingly brilliant (Fig. 52). They are even impressive as they appear as white spots in a dark field, as reproduced in the halftone cut, but this figure is far from satisfactory to illustrate the startling appearance of these fields as observed in the original microscopic examination, or even as shown in kodachrome photomicrographs used as lantern slides or film slides.

Illustrations of chronic tuberculoid pneumoconiosis that were originally inserted in this text under the section devoted to that subject are now inserted just after the illustrations of acute tuberculoid pneumo-



Fig. 51. These small, irregularly spherical masses have the microscopic characteristics of the tubercle of acid-fast bacillus origin, but when they were stained for acid-fast bacilli, none were found but (cont. in Fig. 52)

coniosis, so as to correlate the acute or rapidly developing tuberculoid lesion with the chronic or slowly developing tuberculoid lesion (Fig. 53). The acute dust tubercles (Fig. 51) are relatively free of collagen deposits, whereas the chronic tuberculoid lesion (Fig. 53) has quantities of collagen laid down as whorls surrounded by dust laden phagocytes. These whorls are singularly free of birefractive flecks, as seen by cross polarization (Fig. 54) compared with the surrounding tissue, composed of dust laden phagocytes.

Social and Economic Problems. The social and economic problems of the acute pneumonic parenchymal type and the acute tuberculoid type of pneumoconiosis are the most serious of any of the types of pneumoconiosis because of their rapid development. Indeed, the man may be dead within

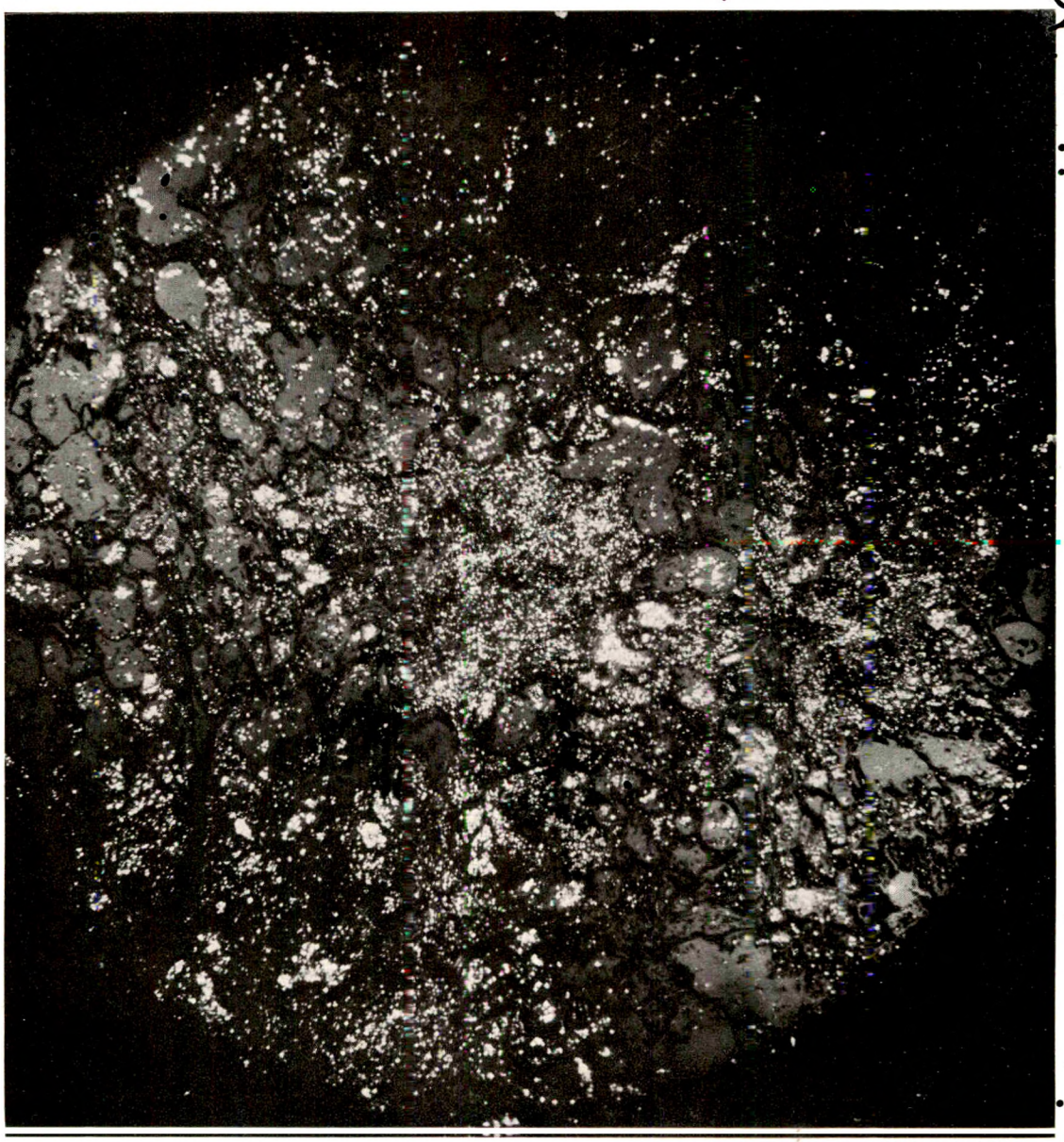


FIG. 52. When the same three tubercles are viewed with a field rendered dark by cross polarization, each tubercle is literally studded with refractive flecks of silica or one of the silicates. This man was dead in less than three years.

two or three years from his first exposure; the patient with the pneumonic parenchymal lesion (Fig. 47) was dead in twenty months; the man with the acute tuberculoid lesion (Fig. 48) was dead in less than three years. These types of lesions are also an important social and economic problem because the roentgen findings are not characteristic of silicosis according to the definition, as they are singularly free of silicotic nodules. For this reason, such lesions may not be recognized as silicosis, and the patient or his family may be deprived of just compensation.

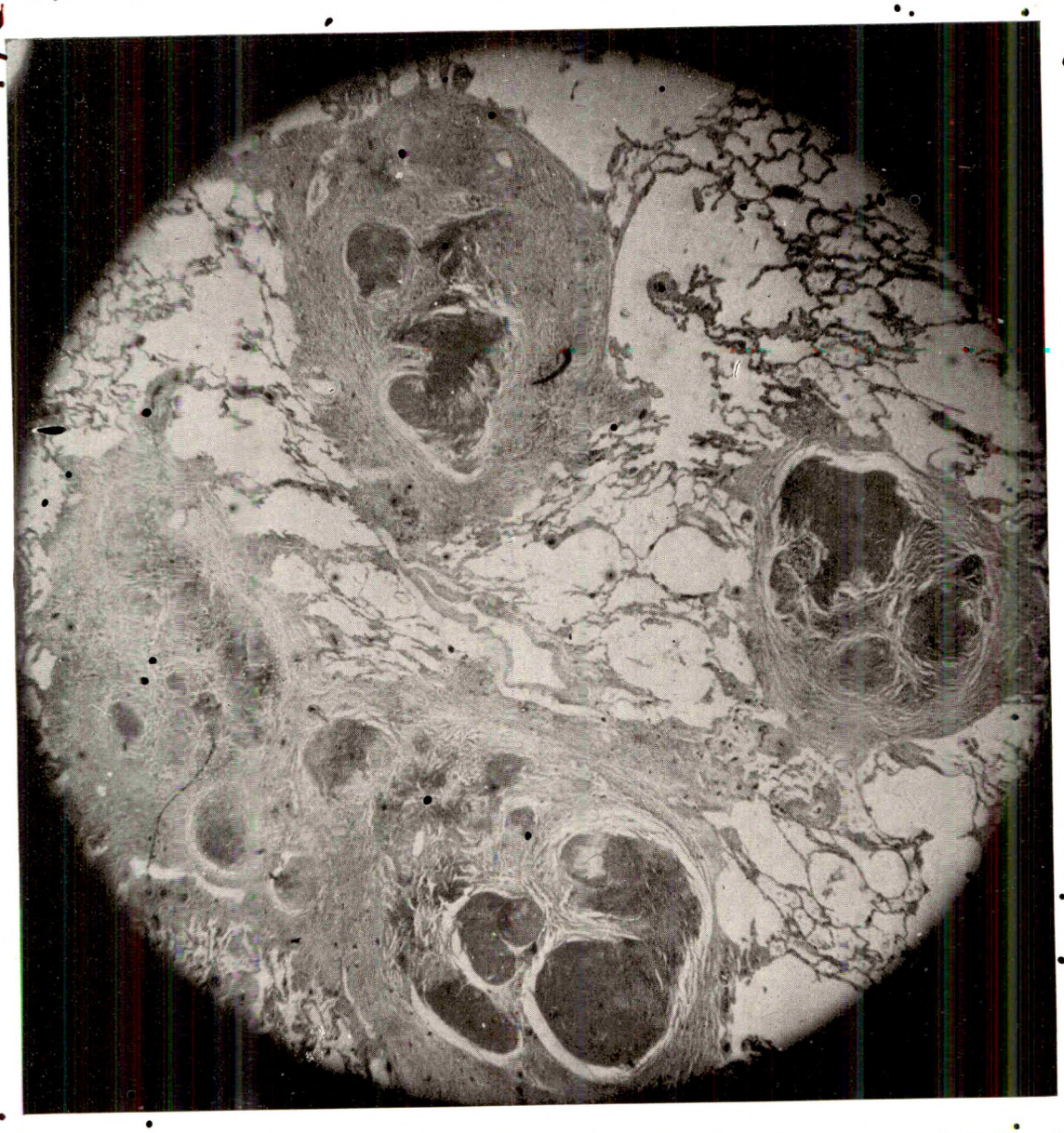


Fig. 53. Likewise, small irregularly spherical masses of long standing have the appearance of old fibrosed tubercles of acid-fast bacillus origin, yet an intensive study by an impartial and unbiased pathologist failed to show any acid-fast bacilli, in a section prepared, cut and stained for this purpose. But when this field was viewed by cross polarization (cont. in Fig. 54)

VASCULAR CHANGES IN PNEUMOCONIOSIS (SILICOSIS)

Under this heading will be considered the morbid changes that occur within the lumen of the pulmonary arteries and veins, and in their walls, exclusive of the laminated deposits of collagen laid down as a sheath around these blood vessels. In an article entitled, "Dyspnea of silicosis: what causes it?" I expressed my conviction that the dyspnea of which the silicotic patient complains is largely due to vascular changes rather than to deposits of connective tissue in the parenchyma or stroma of the lung,

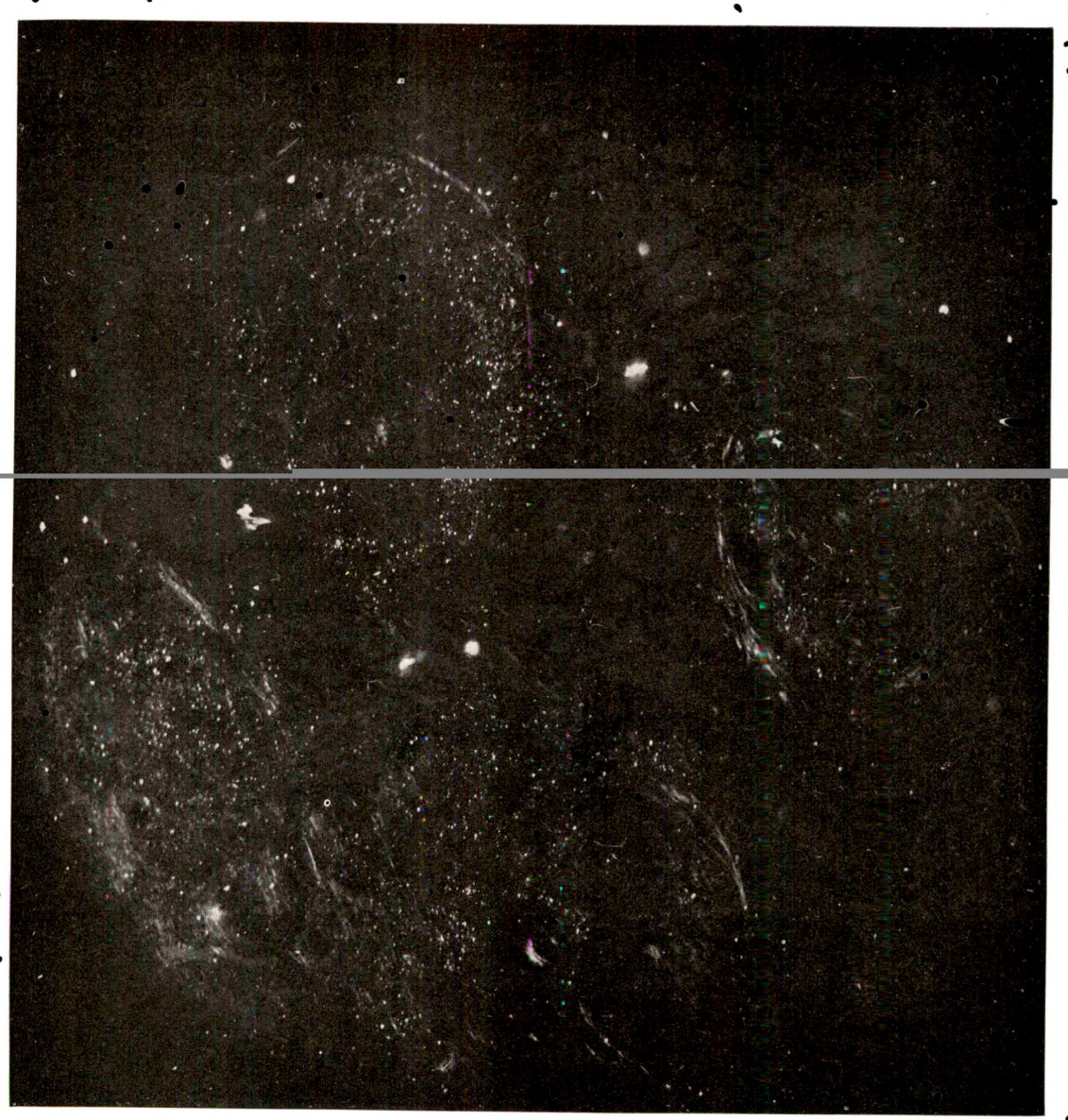


FIG. 54. Quantities of birefractive flecks were discernible. This man had worked for forty years as a granite cutter without a mask and without a blower.

and after four or five years or more intensive study and obtaining very much more data on this subject, I am even more convinced than at the time of presenting the original article. That communication was based upon a study of the acute manifestation of the disease, but subsequent observations have shown even more marked vascular changes to exist in practically all of the microscopic material which I have studied. In the acute cases the vascular changes within the blood vessels were apparently due largely to a slowing down of the circulation of the blood within the avascular areas that was caused by a partial or complete obstruction of the capillary circulation within these avascular areas, with compensating dilatation of the capillaries in the adjacent areas. But the lesions within the lumen of the blood vessel in this

chronic or long standing cases is evidenced by an actual growth of connective tissue within the lumen of the blood vessel, often diminishing its lumen to less than a quarter of its normal diameter (Fig. 55). These connective tissue cells have the aging characteristics typical of connective tissue in that at first they are large, irregularly shaped cells with large nuclei which as they age become fusiform, and still, as they become old, develop into long cylindrical cells with relatively small nuclei. This is one of the most constant findings observed in all of the pathologic material which I have studied.

These vascular changes are very similar to those occurring in coronary occlusion.



FIG. 55. Intravascular growth of reparative connective tissue with cells having aging characteristics have reduced the lumen of the artery to about one-quarter of its normal size. Within the constricted lumen are seen red blood corpuscles and leukocytes. The artery is surrounded by a sheath of collagen, distinctly different from the connective tissue within the artery. An adjacent bronchus surrounded by a sheath of dust laden phagocytes appears in the upper part of the field.



FIG. 56. Roentgenogram of the right lung of a living man with a pulmonary artery injected with diodrast. The roentgenograms were made by Drs. Robb and Steinberg, and were lent to me to show the size, shape and distribution of the pulmonary artery. This roentgenogram was used to make the schematic tracing shown in Figure 20, and it is now compared with (cont. in Fig. 57).

The sudden dyspnea that often occurs late in the course of the disease is similar to coronary attacks. This vascular occlusion or diminution is observed so universally in our pathologic material that it led us to seek to demonstrate the same findings in the living. This thesis was supported by a

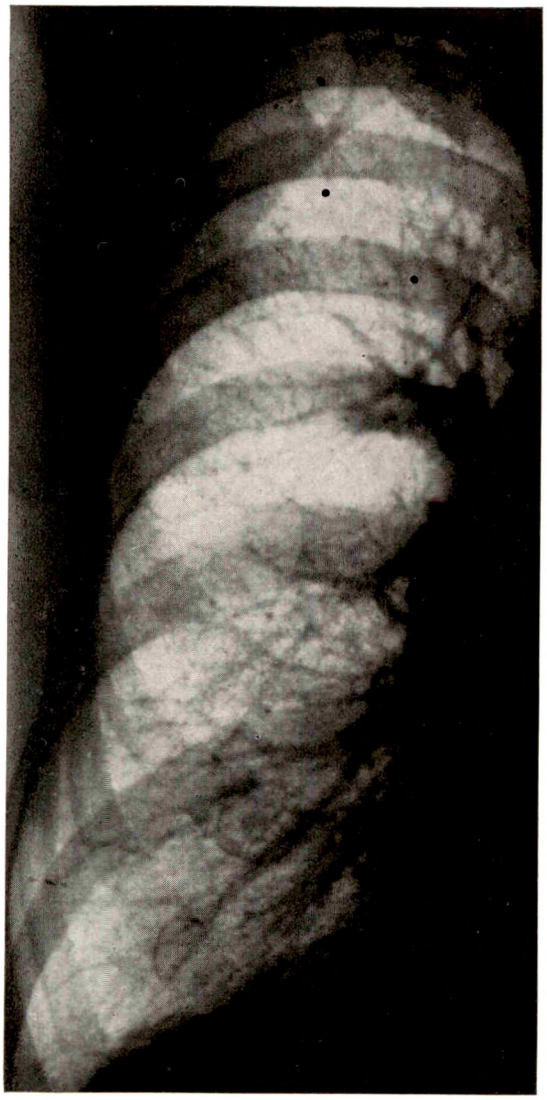


FIG. 57. A roentgenogram of a patient with a history of fifty-six years' exposure to dust, complaining of and actually suffering from a moderate degree of dyspnea. Because of the microscopic findings illustrated in Figure 55, an attempt was made to determine whether the diminution in the lumen of the pulmonary arteries could be shown by vasculograms of the pulmonary circulation. Dr Robb injected the pulmonary circulation in the same manner as in the previous roentgenogram. Various preliminary tests showed a prolonged delay in the diodrast reaching the pulmonary artery, and this finding was amply confirmed by the very marked diminution in the lumen of these arteries, as observed in this roentgenogram, compared with Figure 56, which is normal.

very careful analysis of the clinical history of subjects, where I was able to take the clinical history personally and look for this factor. In one case referred to me by the Mine Workers Union, the clinical history supported this thesis so completely that with the aid of Dr. Robb, I was able to make vasculograms (Fig. 57) of this man showing both the arterial and venous systems of the pulmonary circulation. These vasculograms and the preceding circulatory tests which were necessary to determine the exact instant at which the exposure should be made revealed definite evidence of pulmonary vascular obstruction or stenosis. Roentgenograms were made of both the arterial and the venous systems and the original roentgenograms of the arterial system showed very definite diminution in the diameter of the pulmonary arteries, as compared with similar roentgenograms of the pulmonary artery in the normal subject (Fig. 56) which had been loaned to me by Drs. Robb and Steinberg, whose work on this subject was so epoch making in the study of the pulmonary vascular system. I believe that these vascular changes are less marked in the typical nodular type of pneumoconiosis (silicosis) where the lungs are studded with small, discrete nodules separated by relatively healthy lung than they are in the vesicular type which is relatively free of nodules but where there are deposits of fine strands of collagen involving the terminal blood vessels and bronchioles.

CONCLUSION

As I have said, these highlights and lowlights are extracts from an extensive manuscript prepared for the second edition of the "Story of Dusty Lungs." These findings were observed and recorded, regardless of their significance, or whether they were old or new. No claims are made for original observations, because I have refrained from searching the literature. Some of the findings are conventional and some have been considered unorthodox.

These findings and conclusions presented as they are in this article will give the con-

mentator and critic an opportunity to prove or disprove them, before they are finally published in book form.

The Officers of the John B. Pierce Foundation and the author will welcome any criticism, either constructive or destructive, so that any facts or conclusions herein presented may be retracted or amplified.

56 Greenridge Ave.,
White Plains, New York.

Revelations

FIG. 36. Worked thirty-two years in a mine; positive sputum at the present time.

FIG. 37. History of forty years' exposure to granite dust; no acid-fast bacilli found after three hours' diligent search.

FIG. 38. No exposure to dust; quantities of acid-fast bacilli with growth on culture and positive guinea pig inoculation.

FIG. 39. Tuberculosis of acid-fast bacilli origin.

FIG. 40. Delafield's drawing of pulmonary tubercles of unknown origin.

FIG. 41. Pulmonary tubercles of dust origin; forty years' exposure to granite dust; no acid-fast bacilli found.

FIG. 42. Pulmonary tubercles of acid-fast bacilli origin; quantities of acid-fast bacilli found microscopically.

FIG. 43. History of forty years' exposure to silica dust; no acid-fast bacilli; quantities of silica found microscopically.

FIG. 49. The acute tubercles are of dust origin (transparent, birefractive dust, mostly talc); no acid-fast bacilli found. These pulmonary tubercles are of the same group as shown in Figures 51 and 52.

FIG. 50. The acute tubercles are of acid-fast bacilli origin; no appreciable dust particles present.

DISCUSSION

DR. WILLIAM M. DOUGHTY, Cincinnati, Ohio. I had intended to give a more thorough discussion of this paper, but I don't believe anyone could do justice to it without considerable time and, since we have long passed the hour of closing, I shall limit mine to a few remarks. The amount of effort Dr. Cole has unquestionably put upon this subject, not only in covering the slide problem, but in working out the pathological conditions that are actually shown in all of these various types of silicosis, is an advantage that I think all of us interested in this subject will appreciate the more we go into the pathology of it. Dr. Cole, as you all know, is not only a roentgenologist, but a well recognized pathologist, and I feel that his contribution to the pathology of silicosis at this time is really one that most of us interested in the subject will greatly appreciate, and we shall all look forward to the book which he is to publish in the near future.

AERO-OTITIS MEDIA*†

A ROENTGENOLOGIC STUDY

By LIEUTENANT J. C. LARKIN, (MC) USNR

WHEN the air pressure in the tympanic cavity and the auditory tube is not equal to the pressure in the external auditory meatus and the posterior nasal pharynx, there is a progressive sensation of fullness and pain. Armstrong and Heim[1] described this clinical entity and suggested the term aero-otitis media. The results of this inequality of pressure is a collapse of the tympanic membrane against the labyrinthic wall of the internal ear and a shift of the auditory ossicles. This is usually followed by congestion and inflammation of these structures, and there may be hemorrhage into the tympanic membrane from injury to the small vessels. Unless the tensor veli palatini and the salpingopharyngeus are contracted and pulled away from the cartilaginous portion of the tube by yawning or swallowing, the pressure gradient will become so great‡ as the plane descends that the muscles are unable to open the auditory tube. When this happens, or if one has a post-nasal congestion and cannot open the auditory tube, the sudden sensation of fullness is followed by severe pain, vertigo, tinnitus, and at times even nausea. Hearing may be lost for a few hours or even days. If the pressure gradient exceeds one-half an atmosphere the tympanic membrane will usually rupture.

In aero-otitis media the otoscopic examination of the tympanic membrane will reveal it to be retracted, with a partial or complete loss of the light reflex. The vessels running along the handle of the malleus and those in the periphery of the drum will be dilated and hemorrhagic. The handle of the malleus is shorter, since its more distal portion has been carried toward the labyrinthic wall, and one is now looking at it from a more oblique angle (Gleason[4]). In severe cases there will also be a fluid line behind the drum.

As aero-otitis media is rather common among the hundreds of thousands now engaged in aviation, and since individuals so affected are usually grounded temporarily, the disease entity becomes important in aviation medicine. Since the external auditory meatus and the tympanic membrane are the only structures that can be studied and examined by means now available, it would be desirable to find some method to examine the tympanic cavity, the ossicles, the auditory tube, and its pharyngeal opening. It was hoped that roentgen examination of these heretofore not examinable structures might give additional information on the etiology and even possibly the treatment of this condition.

TECHNIQUE OF ROENTGEN EXAMINATION

A modified vertico-submental view is used, similar to the one used in the routine examination for the sphenoidal sinus (Fig. 1). The patient is placed in the position shown in Figure 2 with the chin extended as far as possible. The 23 degree angle board is used to project the structures posterior to the external canal on the bottom of the film. By the use of a 3 mm. aluminum filter to increase the effective wave length, and a 60 cm. cone with a 10 cm. aperture to cut down the scattered rays, the minute structures appear much sharper and detail that would otherwise be blurred is added to the roentgenogram.

ANATOMY

The lateral portion of the external meatus can be visualized on almost any roentgenogram, but the most mesial portion

‡ 8 cm. of mercury.

* From the Department of Radiology, Dispensary, Naval Air Station, Alameda, California.

† The opinions and views set forth in this article are those of the writer and are not to be considered as reflecting those of the Navy Department.

membrane cuts under the point where the stapes attaches itself to the fenestra vestibuli of the inner ear. This is shown by the broken line of Figure 4. Fortunately this gives an background for the stapes to be visualized on the roentgenogram. In the routine position (Fig. 1) the long axis of the ossicles are perpendicular to the film, so

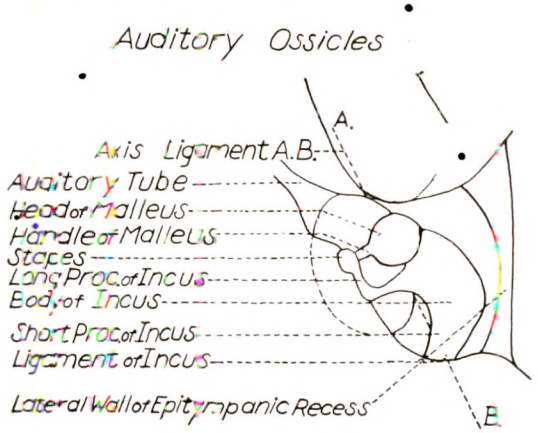


FIG. 4. Drawing to show the relation between the ossicles and the tympanic membrane and also the partition between the middle ear and the external canal.

there is the greatest possible density of bone to absorb the roentgen rays. The head of the malleus and a portion of its neck are almost completely on end so that they cast the densest shadow (Fig. 5). Projecting out from the head, running mesially and posteriorly at an angle of 45 degrees, is the handle of the malleus. The body of the incus is much less dense than the head of the malleus, but casts a shadow several times larger. It is attached to the head of the malleus and has a short and a long process. The short process, the most lateral portion of the ossicle structures, is attached through the ligament of the incus to the wall of the rigid epitympanic recess to form the posterior portion of the axis ligament (Cunningham[3]). The anterior portion of the axis ligament is formed by the anterior ligament of the malleus. The rigid ossicles on rotating about this axis, *AB*, carries the impulses from the tympanic membrane to the stapes. The stapes can be seen on some roentgenograms as a minute projection over the tympanic membrane from the labyrinthic wall of the inner ear. It is the most mesial structure seen, and is situated above the handle of the malleus. On the roentgenogram, however, they appear to be connected. The structure seen just lateral to the ossicles is the lateral wall of



FIG. 5. The auditory ossicles of the ear enlarged about six times. Drawing of Figure 4 is made from this roentgenogram.

the epitympanic recess, and it seems to actually surround the outer half of the ossicles. The dark area mesial to the ossicles is the tympanic cavity and the beginning of the auditory tube.

The bony portion of the auditory tube beginning with the middle ear has a rigid wall, measures about 1.5 cm. long, and can always be seen on the roentgenogram. The cartilaginous portion of the tube is 2.5 cm. long. Its superior and mesial surfaces are rigid, while surfaces on the inferior and lateral sides are made up of the levator veli palatini and the tensor veli palatini respectively. The auditory tube then runs in

is very indefinite. It is difficult to determine where the external canal ends and the middle ear begins since the tympanic membrane cannot be distinguished on the roentgenogram. It can be determined, however, by injecting lipiodol into the external canal (Fig. 3). The medial end of the external canal forms half an ellipse, with the long axis minus 20 degrees from the perpendicular; this is the line formed by the attachment of the tympanic membrane to the bottom of the external canal. By tracing the outline on a clear piece of film, and by drawing in the other half of the ellipse to indicate its superior and lateral portion the attachment of the entire tympanic membrane is determined. Then by superimposing this ellipse over a film showing the ossicles of the same patient, the exact relation between the ossicles and the drum is determined. Figure 4 is copied from such a drawing and shows the desired relationships. It is found that the ossicles of the ear lie above and lateral to the central portion of the tympanic membrane, and only the handle of the malleus and the long process of the incus actually extend over the central portion of the drum. The tympanic

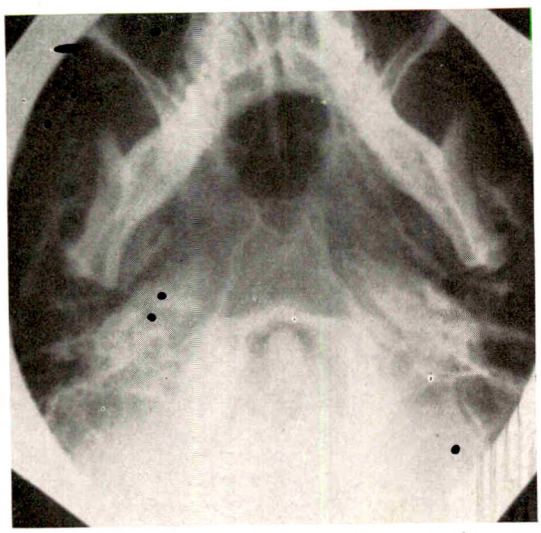


FIG. 1. The routine position for demonstration of the sphenoid sinus. The ossicles, auditory tube, and the pharyngeal ostium can be seen on the left side.



FIG. 2. Position for taking the vertico-submental view. The central longitudinal axis of the cone passes in a line just anterior to the external auditory meatus and posterior to the angle of the jaw.

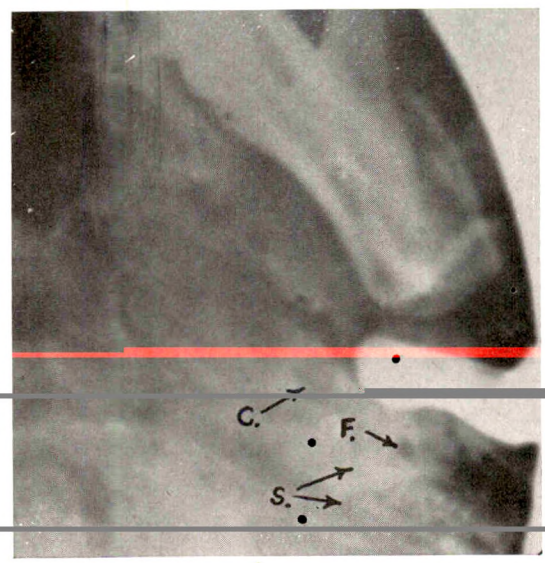


FIG. 3. Lipiodol in the canal to outline the medial aspect of the external auditory meatus. Note the cochlea, *C*, with its three turns. The canal for the facial nerve, *F*, is also seen. This demarcates the region of the semicircular canals, *S*.



Fig. 6. The course of the auditory tube. Note the funnel-shaped opening of the auditory tube in the posterior oral pharynx.

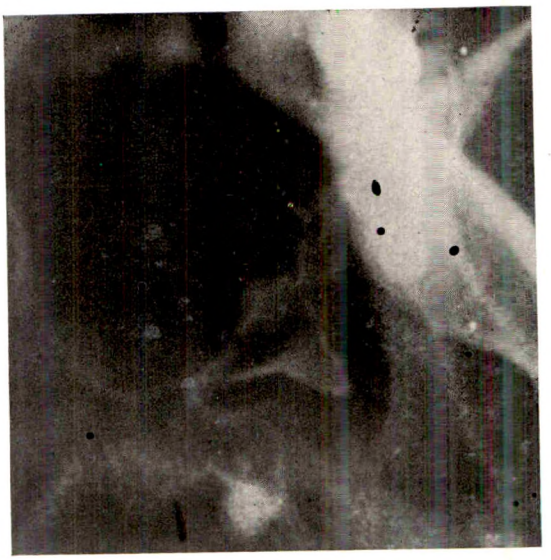


Fig. 7. Lipiodol in the pharyngeal ostium of the auditory tube. The posterior oral pharynx and the membranes have shrunk down on this side.

a direction of about 45 degrees to the posterior nasopharynx, in front of the foramen lacerum and behind the foramen spinosum and ovale. This cartilaginous part of the tube runs parallel to the groove between the greater wing of the sphenoid and the petrous portion of the temporal bones. In some cases the separation is so wide a sphenoid-petrous fissure is formed. But in any case there may be a narrow line on the

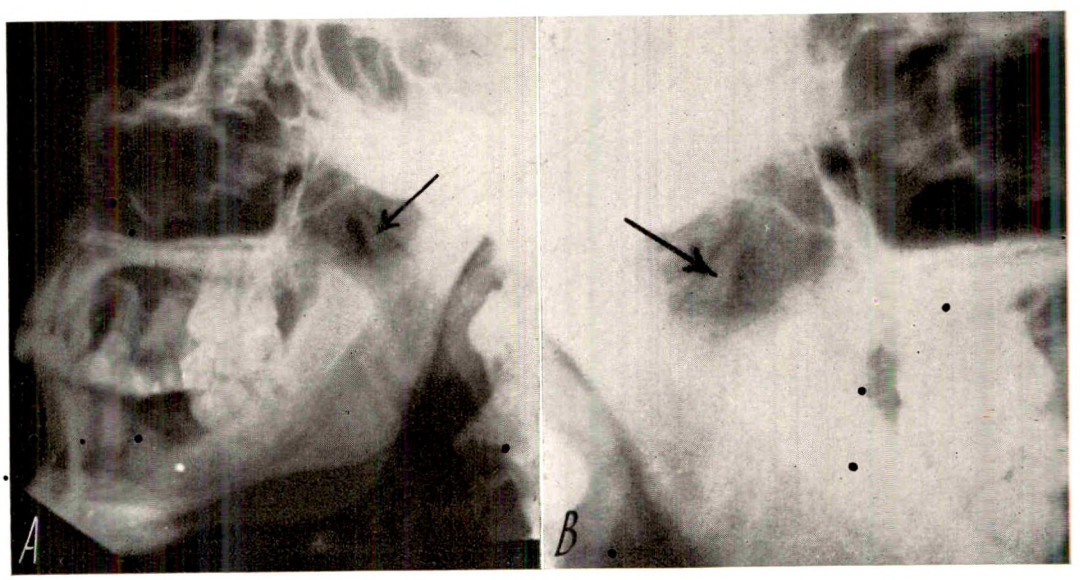


Fig. 8. Pharyngeal ostium of the auditory tube as seen in the lateral view. The opening can be seen best when the right and the left ostia are superimposed.

roentgenogram which may easily be mistaken for the auditory tube. However, the tube is usually wider and more lateral, and there can be no mistake in its identity when it is seen to end in the funnel-shaped pharyngeal ostium (Fig. 6). The junction between the cartilaginous and bony portions unfortunately is obscured by the spine of the sphenoid bone. The part that enters the posterior oral pharynx runs beneath the medial pterygoid plate, and therefore it also cannot always be seen. Between these two obscure points, indicated by arrow in Figure 11, Case 1, is a shadow measuring 2 mm. wide and 20 mm. long which is the cartilaginous portion of the auditory tube distinguishable on the roentgenogram and called the "Tubenknorpel" (Corning[2]).

The exact position of the pharyngeal ostium of the auditory tube and its shape and contour are determined by placing lipiodol in the ostium (Fig. 7). The ostium is also seen in the routine lateral view taken to show the soft tissues of the neck (Fig. 8). It is necessary to cocainize the posterior nasopharynx to insert the cannula, and in so doing there is shrinking of the mucous membranes. The ostium is actually a vertical slit, but when seen from above (Fig. 7) it appears to be funnel shaped. The lateral border of the ostium in Figure 7 is the superior portion as seen in Figure 8, and the mesial border is the inferior portion seen in the lateral view. The auditory ostium is located on each side of the posterior nasopharynx, just anterior to the fossa of Rosenmüller and posterior to the inferior nasal meatus.

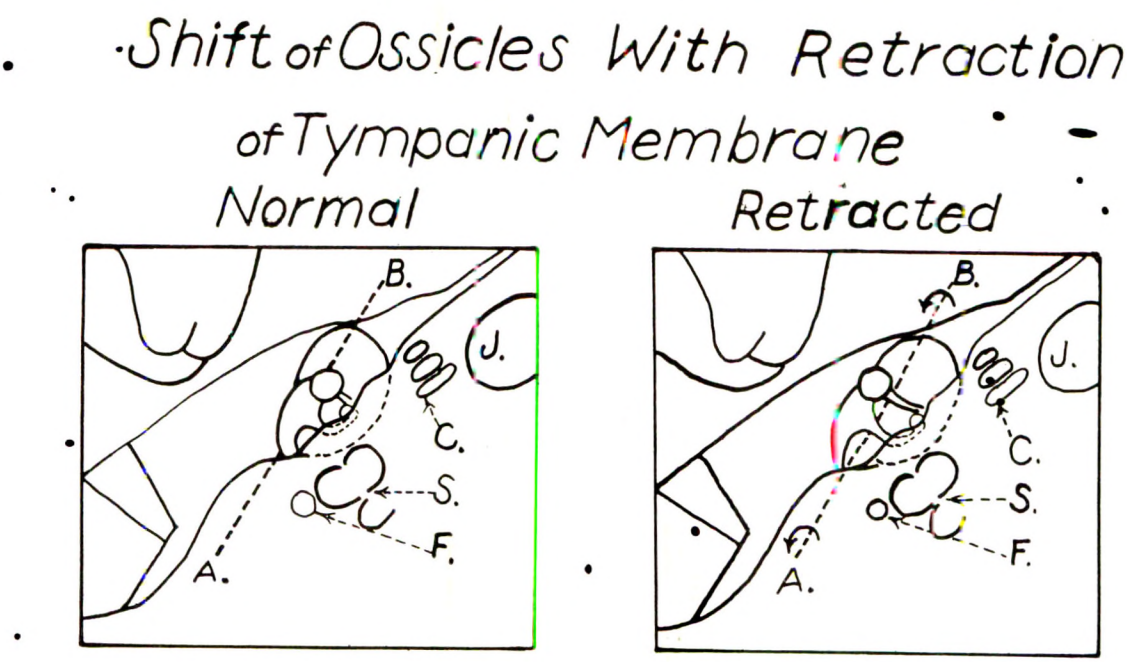


FIG. 9. Drawing of the auditory ossicles when the tympanic membrane is in normal position. *C*, the cochlea; *F*, the facial canal; *S*, the semicircular canals. These structures are seen so seldom on roentgenograms that neither their presence nor their absence has any diagnostic significance.

FIG. 10. The position of the ossicles after retraction of the tympanic membrane. The ossicles have rotated about the axis, *A B*, and have assumed a new position more anterior and more lateral than is seen in Figure 9

POSITION OF THE TYMPANIC MEMBRANE

The auditory ossicles are opaque, and since they move with the tympanic membrane, it is possible to determine the position of the membrane indirectly by the

position assumed by the ossicles. Figure 9 shows the axis, *AB*, and the position assumed by the ossicles in relation to the other structures. When the handle of the malleus is carried inward by the retraction of the tympanic membrane, the rigid ossicles must also move; but since they rotate about the axis *AB*, the head of the malleus and the parts of the ossicles above the axis must move in a direction just opposite to that of the handle. It then assumes a position seen in Figures 10 and 15, Case v. It is noted the distance between the head of the malleus and the stapes is increased, and as a result the handle of the malleus appears longer in Figure 10 than in Figure 9. Also, when this happens, since the axis *AB*, is at an oblique angle to the auditory canal, the ossicles must also move across the tympanic cavity to a new position that is both more anterior and also more lateral, as is shown in the drawings.

PRESENCE OF FLUID IN THE MIDDLE EAR

This condition, known as hydrops ex vacuo, is thought to result from the creation of partial vacuum in the ear, causing a change in the osmotic relations and resulting in a flow of lymph into the middle ear. This can at times be seen as a fluid line upon examination of the drum with the otoscope. The ossicles are, however, very often found to be cloudy on the roentgenograms, since only a small amount of exudate is needed to obscure their outlines. Thus small amounts of fluid can be detected which would be difficult to find by other methods.

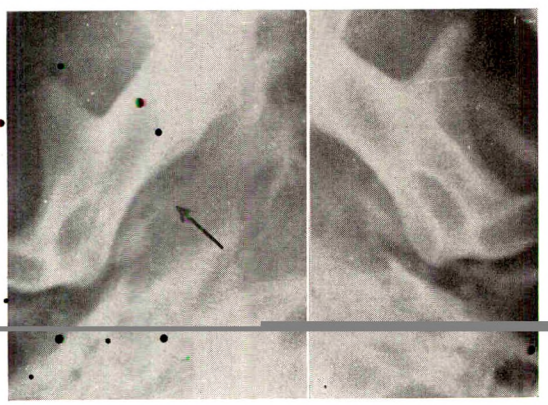


Fig. 11. On the left side the ossicles and surrounding air cells are cloudy, indicating aero-otitis media. The head of the malleus has assumed a more lateral and anterior position. The cartilaginous part of the tube is quite prominent on the injured side.

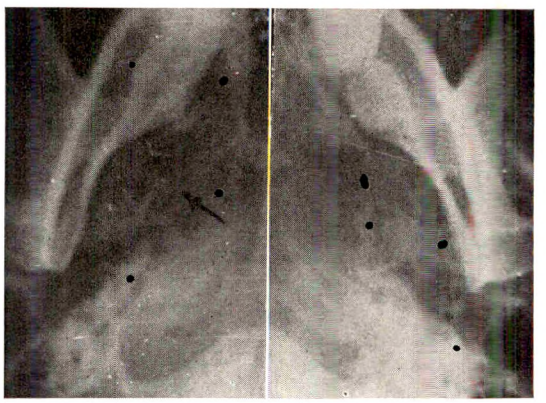


Fig. 12. The hazy ossicles and surrounding air cells on the right side together with the shift of the ossicles indicates an aero-otitis media. On the left side there is also a slight shift, but there is no indication of fluid and congestion.

CLINICAL APPLICATIONS

Methods have now been worked out for the examination of the middle ear, the position of the tympanic membrane and its relation to the ossicles of the ear, the determination of fluid in the middle ear, and the patency of the auditory tube. These methods of examination are now applied to some of the conditions found on examination of flyers suffering from injuries to their auditory mechanisms. It is also possible to study the effect of drugs on these structures.

CASE HISTORIES

Case I (Fig. 11). This dive-bomber pilot received his injury two weeks ago. The right side has completely recovered but he still has a feeling of fullness in the left ear.

Case II (Fig. 12). A dive-bomber pilot, aged twenty-two, who on a dive-bomber mission developed bilateral blockage on his first dive from 14,000 feet. He was able, however, to get air into the left auditory tube, but the right remained blocked. On examination the right

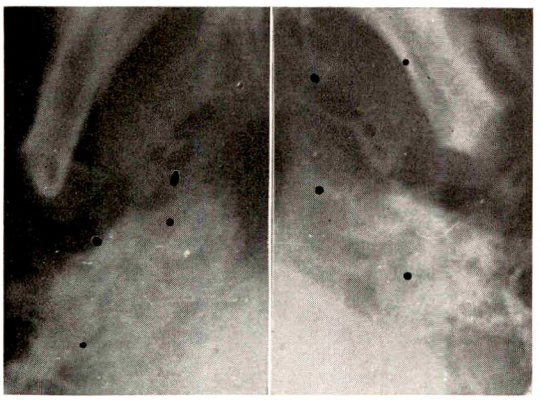


FIG. 13. The head of the malleus has shifted almost to the lateral wall of the epitympanic recess, and there is a small amount of clouding on the right side. The stapes is unusually prominent Left side is normal.

drum was scarlet, and the left appeared to be normal.

CASE III (Fig. 13). A transport pilot who on descending developed bilateral blockage. On examination the left drum is normal. The right shows the typical appearance of aero-otitis media.

CASE IV (Fig. 14). A dive-bomber pilot developed bilateral aero-otitis media after completing his mission two hours before. Most of the dives were made from a height of 15,000 feet. He has had chronic aero-otitis media for the past two years.

CASE V (Fig. 15). An A.M.M. 3/c developed pain in the right ear while in the low pressure

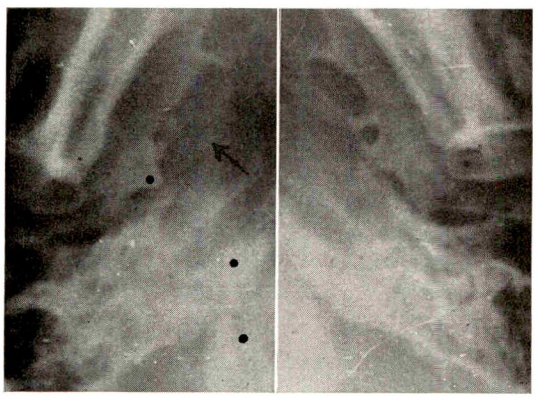


FIG. 14. The ossicles are obliterated on both sides. The cartilaginous part of the tube is well visualized on the left side.

chamber on being returned to normal a pheric pressure. The air had been reduce pressure corresponding to that found height of 35,000 feet.* He was able to flate his left auditory tube, but the rig mained blocked.

CASE VI (Fig. 16). An S. 2/c developed

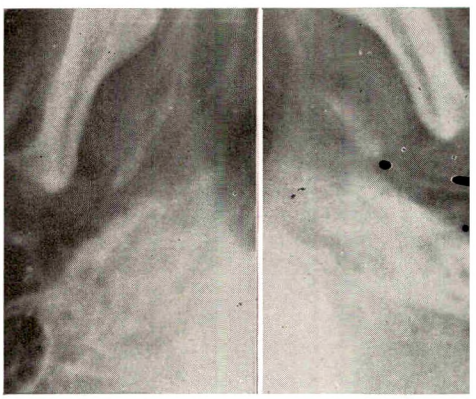


FIG. 15. The right side shows a shift of the c similar to that in drawing of Figure 10, w the normal left side the ossicles are similar t of drawing, Figure 9. There is also slight cl on the injured right side.

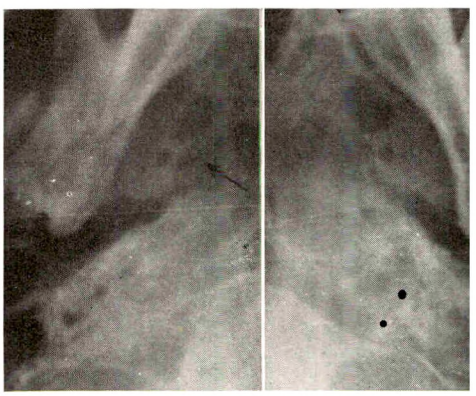


FIG. 16. The ossicles are cloudy and shifted la on the left side, and the auditory tube is nent. The right side is normal.

otitis media after an indoctrination run low pressure chamber. All his symptoms the right side, and the left side is asy matic.

* The incidence of aero-otitis media is quite high in m indoctrinated in flying activities in low pressure chambe

DISCUSSION

Unfortunately the auditory structures are not always demonstrable on the roentgenograms, so that their presence or absence is of no clinical value; and it is only when the right and left sides can be compared that the examination yields useful information. The finding of abnormally placed ossicles in cases having no symptoms suggests that position is not an absolute criterion for symptomatic aero-otitis media. However, the blurring of the ossicles due to congestion and the accumulation of exudate in the tympanic cavity and the surrounding air cells show correlation with the clinical findings. This roentgen study reveals for the first time the presence of fluid in the cells of the petrous bone and in the cells of the mastoid region in cases of aero-otitis media. This is due to hydrops ex vacuo and is a result of fluid oozing from the mucous membrane lining the air cells and covering the auditory ossicles. The presence of this fluid in the air cells probably accounts for the sensation of fullness experienced.

When the tensor palatini contracts it changes the normally collapsed auditory tube into one with a wide lumen. Since it is common to find the shadow of the "Tubenknorpel" on one side only (Case 1, Fig. 11), and since the two sides are identical anatomically, the unilateral shadow on the roentgenogram must be due to air in the lumen. Thus this part of the auditory tube could not prevent the passage of air into the tympanic cavity. Three cases of aero-otitis media reported here do have such tubes, on the affected side, and hence the constriction must be at the ostium.

SUMMARY

Methods have been worked out for the roentgen examination of the external auditory meatus, the shifting of the tympanic membrane as shown by the position assumed by the auditory ossicles, the presence of fluid in and about the tympanic cavity, and the patency of the auditory tube.

The above methods have been applied to a number of cases of aero-otitis media in an attempt to study the mechanism of the injury and to find a possible remedy.

It is found that the position of the auditory ossicles is not an absolute criterion for the diagnosis of aero-otitis media. However, the clouding of the auditory ossicles and of the surrounding cells in the petrous bone and mastoid areas due to the presence of fluid indicates aero-otitis media.

In aero-otitis media the auditory tube is probably blocked, usually in the pharyngeal ostium.

The use of 10 per cent cocaine causes a marked shrinking of the mucous membranes of the posterior oral pharynx and of the pharyngeal ostium. This can be demonstrated on the roentgenogram.

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THE EFFECT OF ROENTGEN RAYS ON THE JOINT EFFUSIONS IN CERTAIN NONSPECIFIC ARTICULAR LESIONS IN HUMANS, AND ON THE NORMAL JOINTS OF DOGS

A PRELIMINARY REPORT

By THOMAS HORWITZ, M.D.,* *and* MELVIN A. DILLMAN, M.D.†
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IT IS NOT claimed, as a result of these clinical and experimental observations, that roentgen therapy is or is not beneficial in the treatment of certain arthritides. It is felt, however, that, in the doses administered, this form of therapy has been valuable *in the control of the joint effusions* found in certain nonspecific synovial lesions involving the knee joints in humans, and that, within the recommended doses, there have been no immediate or as yet remote deleterious effects. The longest observation period in this group has been from October 12, 1940, to the present (February, 1942). The more vulnerable articulations of the extremities of dogs, treated with much larger doses than have been utilized and found effectual in human subjects, have shown no immediate and as yet no remote ill effects on the articular and para-articular structures. The longest observation period in the animals has been from December 16, 1940 to the present (February, 1942). No attempt has been made to duplicate, in animals, the nonspecific synovial lesions encountered in the human subjects, because of the absence of known specific criteria as to the etiology and nature of these articular lesions.

USE OF ROENTGEN THERAPY IN THE CONTROL OF THE JOINT EFFUSIONS IN CERTAIN NONSPECIFIC SYNOVIAL LESIONS INVOLVING THE KNEE JOINTS IN HUMANS

One knee joint in 3 subjects and both knee joints in 3 others were treated—a total of 9 knee joints. In these 6 cases no other joints than the knees were involved. In 2 patients the effusions were intermittent. In 1 subject (F.C.) the phenomenon occurred in one knee, and, after eleven months of recovery in this knee following roentgen therapy, appeared in the opposite knee, and in the other patient (S.G.) the intermittent effusion occurred in only one knee. In the interval periods between attacks, the involved knees in these 2 cases were anatomically and functionally normal, and asymptomatic. In 3 patients the synovial effusion was constantly present—in 1 subject (W.M.) occurring in one knee and appearing, five months after recovery of that knee following roentgen therapy, in the opposite knee, and in the other patients (M.D. and J.B.) the knee involvement being unilateral. In the youngest patient (F.S.), there were continuous effusions present simultaneously in both knees. In the 4 cases with constant knee effusions, aspirations of the affected joints restored, temporarily, a full range of painless motion, but revealed some residual thickening of the joint lining in 3 subjects. The only complaint in our 6 cases was that of knee effusion, with incidental "sense of fullness" in the joint and impaired range of motion. Extensive clinical laboratory examinations—including urinalyses; blood count, blood sedimentation rate, blood Wassermann and Kahn tests and complete blood chemistry estimations; cytologic, bacteriologic, serologic and streptococcus agglutination studies of the joint fluid; intracutaneous tuberculin test and

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roentgenographic studies—were uniformly normal or negative.

The etiology could not be definitely ascertained, the onset being gradual and without apparent cause in all of the subjects, with the possible exception of the young patient (F.S.) who had suffered an attack of rheumatic fever prior to the onset of her knee involvement. The diagnosis in the 2 cases with recurrent knee effusions was "intermittent hydrarthrosis" and in the 4 cases with continuous effusions was "chronic nonspecific synovitis with effusion." In all cases, previous treatment by repeated aspirations, compression bandages, external fixation and intensive physiotherapy, and, in 2 cases, nonspecific protein therapy, had failed to alter the clinical picture.

The methods of treatment and the results in these cases are given in Table 1.

Synovial biopsy, under local anesthesia, was performed through a 2 inch linear anteromedial incision, prior to or during roentgen therapy in both knees of 2 cases (F.C. and W.M.), and in one knee prior to treatment in a third case (M.D.), in an effort to determine the nature of the synovial lesions and to ascertain the modus operandi of roentgen therapy in the control of the joint effusions. The slight variations in the technique of deep roentgen therapy were due to the necessity of our employing two different machines during the investigation.

Results of Roentgen Therapy in the Human Subjects.

Clinical. There were no untoward reactions evident under the fractionated therapy, even in those receiving two full courses of roentgen therapy to the knee joint, except in 1 case (J.B.) where a slight erythema was observed after the first course had almost been completed (1,296 roentgens to the inner and 1,296 roentgens to the outer aspects of the knee). The effusion in this case failed to respond to this series, and the patient did not return for further treatment. The 8 knee joints treated in the other 5 patients showed favorable responses during the first or second course of roentgen therapy. The full effect was observed in 7 of the 8 knee joints at or just prior to the termination of the full course. In the right knee of W.M., complete recovery was not observed until one month after the completion of the course of roentgen therapy, suggesting that in this instance some benefit may have accrued from cumulative roentgen-ray effect. In the right knee of F.C. only a slight response followed one course of roentgen treatment, but complete recovery followed a biopsy procedure without further roentgen therapy. Although this might suggest a possible beneficial effect of the arthrotomy *per se*, the evidence is otherwise in the other instances where a biopsy was performed. Its use *prior* to roentgen therapy was without apparent clinical benefit in the left knee of the same patient, in the right knee of W.M., and in another case with chronic effusion of one knee, not herein reported, who failed to return for roentgen therapy.

In F.C., there has been complete recovery from recurrent effusions in both knees that are, at present, asymptomatic and anatomically and functionally normal. The same result has been obtained in W.M., whose knees were the site of chronic joint effusions. In one knee in M.D. and in both knees in F.S., the constant synovial effusions have disappeared and the joints are asymptomatic, although there is some residual synovial thickening in each instance. In S.G., although the distressing recurrent effusions no longer occur in the left knee, there is some residual synovial thickening and discomfort on weight bearing.

Roentgenographic. There has been no evidence, thus far, on repeated roentgenographic examinations, of change in the osseous and para-osseous structures of the treated and control knee joints. The longest period of observation has been from October 12, 1940 to the present (February, 1942).

TABLE I

ROENTGEN THERAPY FOR CERTAIN NONSPECIFIC SYNOVIAL LESIONS OF THE KNEE JOINT IN HUMANS

Case	Sex	Age	Knee Joint Involved	Nature of Effusion	Duration	Date Treatment Begun	Time of Biopsy in Relation to Therapy	Factors in Roentgen Therapy (two fields: inner and outer surfaces of knee)	Total Dose (roentgens)	Results to Date
F.C.	F	54	Left	Intermittent. Every 10 days and lasting 4 days	27 yr.	Oct. 16, 1940	Before	Kv. 200; ma. 6; skin target distance 50 cm.; filter 0.5 mm. copper and 1 mm. aluminum; time 11 min.; each dose 198 r; 7 weekly treatments to both fields	1,386 to each field	No effusions and complete anatomical and functional recovery, since Nov., 1940 or 3 wk. prior to termination of roentgen therapy
			Right	Intermittent. Every 10 days and lasting 4 days	3 wk.	Sept. 23, 1941	After	Kv. 200; ma. 6; skin target distance 50 cm.; filter 0.5 mm. copper and 1 mm. aluminum; time 5 min.; each dose 215 r; 6 weekly treatments to both fields	1,290 to each field	Only partial diminution in severity of intermittent effusions following roentgen therapy; complete recovery following biopsy, Dec., 1941
S.G.	F	22	Left	Intermittent. Every 2 wk. and lasting 6 days	5 yr.	June 17, 1941	None	Course 1: kv. 200; ma. 6; skin target distance 50 cm.; filter 0.5 mm. copper and 1 mm. aluminum; time 11 min.; each dose 198 r; 4 weekly treatments to inner and 3 weekly treatments to outer fields. Followed by: kv. 200; ma. 25; skin target distance 50 cm.; filter 0.5 mm. copper and 1 mm. aluminum; time 5 min.; each dose 215 r; 3 weekly treatments to inner and 4 weekly treatments to outer fields. Treated biweekly, but only once weekly to each field, for 7 weeks	Course 1: 1,437 to inner and 1,455 to outer fields	Complete recovery from intermittent effusions. Residual pain and slight synovial thickening (since Nov., 1941)
								Course 2: kv. 200; ma. 25; skin target distance 50 cm.; filter 0.5 mm. copper and 1 mm. aluminum; time 5 min.; each dose 215 r. No. of treatments as in Course 1. Interval between two courses, 10 weeks	Course 2: 1,505 to each field	
W.M.	M	51	Right	Continuous	1 yr.	Oct. 12, 1940	Before Course 1	Course 1: kv. 200; ma. 6; skin target distance 50 cm.; filter 0.5 mm. copper and 1 mm. aluminum; time 11 min.; each dose 198 r; treated biweekly, once weekly to each field, for 7 weeks	Course 1: 1,386 to each field	No effusion and complete anatomical and functional recovery, since April, 1941 or one month after termination of Course 2
								Course 2: as Course 1. Interval between two courses, 6 weeks	Course 2: 1,386 to each field	
			Left	Continuous	2 wk.	Aug. 12, 1941	Following Course 1 and preceding Course 2	Course 1: kv. 190; ma. 25; skin target distance 50 cm.; filter 0.5 mm. copper and 1 mm. aluminum; time 5 min.; each dose 215 r; treated biweekly, once weekly to each field, for 7 weeks	Course 1: 1 505 to each field	No effusion and complete anatomical and functional recovery since termination of Course 2 in Jan., 1942
								Course 2: as Course 1. Interval between two courses, 6 weeks	Course 2: 1,505 to each field	
M.D.	F	58	Right	Continuous	1 yr.	June 17, 1941	Before Course 1	Course 1: as Course 1 in Case S.G.	Course 1: 1,437 to inner and 1,455 to outer fields	Disappearance of all effusion at termination of Course 2 in Nov., 1941. Slight residual synovial thickening
								Course 2: as Course 1 in Case S.G.	Course 2: 1,505 to each field	
J.B.	M	31	Right	Continuous	9 mo.	Nov. 18, 1941	None	Kv. 200; ma. 25; skin target distance 50 cm.; filter 0.5 mm. copper and 1 mm. aluminum; time 5 min.; each dose 215 r; biweekly treatments, once weekly to each field, for 6 weeks	1,296 to each field	No diminution of effusion. Single course not completed due to slight erythema. Did not return for second course
F.S.	F	13	Bilateral and Simultaneous	Continuous	4 yr.	Oct. 21, 1941	None	Course 1: kv. 200; ma. 25; skin target distance 50 cm.; filter 0.5 mm. copper and 1 mm. aluminum; time 5 min.; each dose 215 r; three biweekly treatments, once weekly to each field of both knees treated simultaneously	Each knee—Course 1: 645 to each field	All effusion disappeared in both knees after termination of Course 2 in Dec., 1941. Residual asymptomatic synovial thickening in both knees
								Course 2: as Course 1. Interval between two courses, 6 weeks	Course 2: 645 to each field	

. It is anticipated, although not as yet evident, that early epiphyseal growth arrest may be initiated in the knee regions of the thirteen year old subject, even with the minimal therapeutic doses used. This will not be considered untoward in this case if it occurs, since the dosage has been administered equally to both knees, and because the child has already equaled the height of her parents.

Histopathologic. Synovial biopsy was performed *prior* to any treatment in 3 cases (M.D., F.C.—left knee; W.M.—right knee). The pathologic features bore no constant relation to the clinical features in these 3 cases and for this reason an attempt to classify the clinicopathologic features in our cases has not appeared worth while. For example, the clinical features of M.D. and W.M. were similar in that there was asymptomatic, continuous swelling of the knee, with normal laboratory and roentgenographic findings, and in each subject recovery followed biopsy and roentgen therapy, except for some residual synovial thickening in M.D. The histopathologic features, however, were not alike. In both biopsy specimens the synovial layer of cells appeared normal, and the pathologic changes were confined to the subsynovial tissues that were edematous and very vascular. In M.D. there was a very diffuse sprinkling of monocytes, lymphocytes, plasma cells and red blood cells throughout the subsynovial connective tissue, while in W.M. there were numerous focal perivascular collections of chronic inflammatory cells. This latter histopathologic picture was present also in F.C., even though in this case the swelling was not constantly present but was regularly intermittent. In F.C. the laboratory and roentgenographic findings were also normal, and the recovery following biopsy and roentgen therapy was complete. Special fat stains in these 3 specimens showed only scattered lipoid deposits.

In 2 cases, synovial biopsy was performed *after* one full course of roentgen therapy (1,290 r to the inner and 1,290 r to the outer sides of the right knee of F.C., and 1,505 r to the inner and 1,505 r to the outer aspects of the left knee of W.M.). In both cases there had already been evidence of improvement. The biopsies, however, were performed to secure histopathologic material for comparison with the specimens removed from the opposite knees of these cases prior to roentgen therapy. The specimens removed *following* roentgen therapy showed the surface synovial cells to be normal. The subsynovial tissues still demonstrated numerous perivascular collections of chronic inflammatory cells, but illustrated *three additional features:* (1) disappearance of edema, (2) a diffuse fibrosis and (3) thickening of the vessel walls with obliteration of the lumina of the smaller vascular channels. There were no areas of tissue degeneration or tissue death. In W.M. there were many large macrophages, filled with phagocytosed blood pigment granules, in the perivascular lymphocytic collections (Fig. 1 and 2).

ROENTGEN THERAPY TO THE NORMAL PERIPHERAL JOINTS OF DOGS

Six dogs, all male hounds about one year old and in apparently perfect health, were utilized in this study. The areas surrounding the treated joints were protected during roentgen therapy and the comparable joints of the opposite limbs were used as untreated controls. The left knee (stifle) and elbow joints were treated in all 6 animals, while in dog No. 3 the right hip was also treated and the left hip used as the untreated control. These dogs were kept in cages and the functional stress on their limbs was kept to a minimum.

An effort was made to study the effects of the following factors which have been evaluated in Table II:

1. The *type* of roentgen therapy—superficial or deep (always fractionated).
2. The *dose* of each roentgen treatment, and the total amount of roentgen-ray dosage.
3. The *duration* of active treatment, the duration of the cumulative period, and the

TABLE II

ROENTGEN THERAPY TO THE NORMAL JOINTS OF DOGS

Dog No.	Weight (lb.)	Date Treatment Begun	Joint Treated	Type of Therapy	Factors of Therapy (single field: medial aspect of joint)	Total Dose (roentgens)	Duration Treatment (weeks)	Duration Cumulative Period (weeks)	Effects		
									Clinical	Roentgenographic	Histopathologic
4	25	Dec. 23, 1940	L. knee (stifle joint)	Superficial	Kv. 135; ma. 5; skin target distance 30 cm.; filter 4 mm. glass; time 4 min.; each dose 200 r, every 2nd day for 15 treatments	3,000	4	8	None	None	None
			L. elbow	Deep	Kv. 200; ma. 6; skin target distance 50 cm.; filter 0.5 mm. copper and 1 mm. aluminum; time 10 min.; each dose 200 r, each 2nd day for 15 treatments	3,000	4	2	None	None	None
1	12	Jan. 11, 1941	L. knee	Superficial	Kv. 135; ma. 5; skin target distance 30 cm.; filter 4 mm. glass; time 10 min.; each dose 500 r, every 2nd day for 12 treatments	6,000	4	4	Loss of hair and superficial ulceration	None	None
			L. elbow	Deep	Kv. 200; ma. 6; skin target distance 50 cm.; filter 0.5 mm. copper and 1 mm. aluminum; time 20 min.; each dose 400 r, every 2nd day for 15 treatments	6,000	4	2	None	None	None
5	20	March 2, 1941	L. knee	Deep	Kv. 200; ma. 6; skin target distance 50 cm.; filter 0.5 mm. copper and 1 mm. aluminum; time 10 min.; each dose 200 r, every 2nd day for 20 treatments	4,000	6	12	None	None	None
			L. elbow	Deep	Kv. 200; ma. 6; skin target distance 50 cm.; filter 0.5 mm. copper and 1 mm. aluminum; time 10 min.; each dose 200 r, every 2nd day for 10 treatments	2,000	3	8	None	None	None
3	14	Feb. 24, 1941	L. knee	Deep	Kv. 200; ma. 6; skin target distance 50 cm.; filter 0.5 mm. copper and 1 mm. aluminum; time 10 min.; each dose 200 r, every 2nd day for 20 treatments during Course 1 and daily for 20 treatments during Course 2	8,000 Course 1—4,000 Course 2—4,000	11 Course 1—6 Course 2—5	8 Between courses—5 Between Course 2 and death—3	None	None	None
			L. elbow	Deep	Above factors; each dose 200 r; two courses of 10 treatments each, administered every 2nd day	4,000 Course 1—2,000 Course 2—2,000	6 Course 1—3 Course 2—3	11 Between courses—8 and death—3	None	None	None
			R. hip	Deep	Above factors, each dose 200 r, one course of 15 treatments, administered every 2nd day	3,000 (only one course)	3	4	None	None	None
2	16	Aug. 4, 1941	L. knee	Deep	Kv. 200; ma. 25; skin target distance 50 cm.; filter 0.5 mm. copper and 1 mm. aluminum; time 5 min.; each dose 200 r, daily for 20 treatments	4,000	3	From Aug. 28, 1941 to Feb., 1942	None	None	Still living
			L. elbow	Deep	Above factors; each dose 200 r, daily for 10 treatments	2,000	10 days	From Aug. 15, 1941 to Feb., 1942	None	None	
6	14	July 17, 1941	L. knee	Deep	Kv. 200; ma. 25; skin target distance 50 cm.; filter 0.5 mm. copper and 1 mm. aluminum; time 10 min.; each dose 400 r, daily for 10 treatments	4,000	10 days	From Sept. 29, 1941 to Feb., 1942	None	None	Still living
			L. elbow	Deep	Above factors; each dose 400 r, daily for 5 treatments	2,000	5 days	From Sept. 29, 1941 to Feb., 1942	None	None	

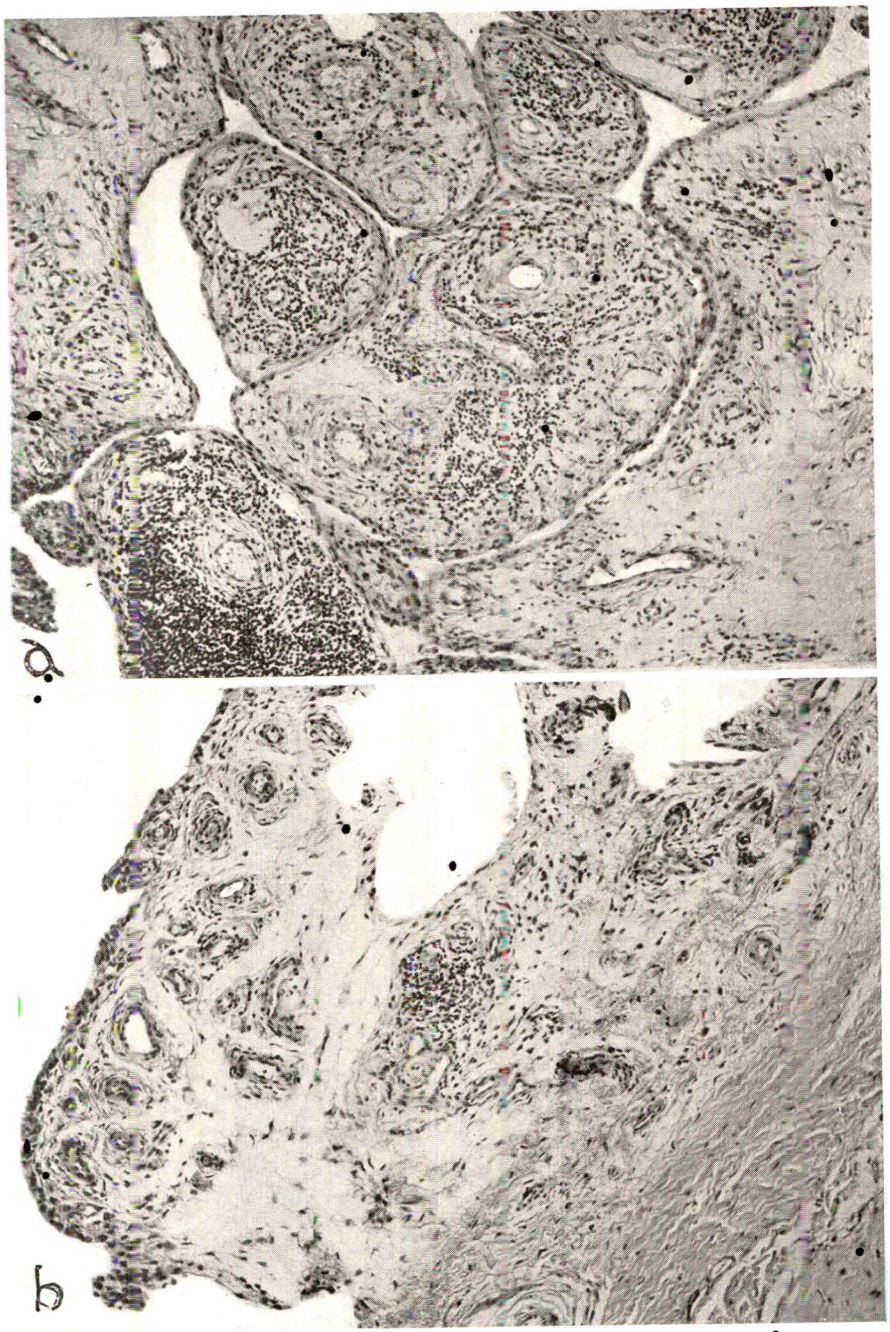


FIG. 9. *a*. Photomicrograph (×110) of synovia of right knee of patient F.C., *prior* to roentgen therapy. The surface cells lining the closely packed synovial villi are normal, but the subsynovial tissues demonstrate considerable edema, many dilated blood vessels and many focal perivascular collections of chronic inflammatory cells.

b. Photomicrograph (×110) of synovia of left knee of the same patient, *following* a course of roentgen therapy (1,290 r to inner and 1,290 r to outer fields). The surface cells show no considerable change. There is less edema of the subsynovial tissues, the vessels are no longer dilated and the lumina of several vessels have become obliterated, while there is only one collection of chronic inflammatory cells in the entire field.

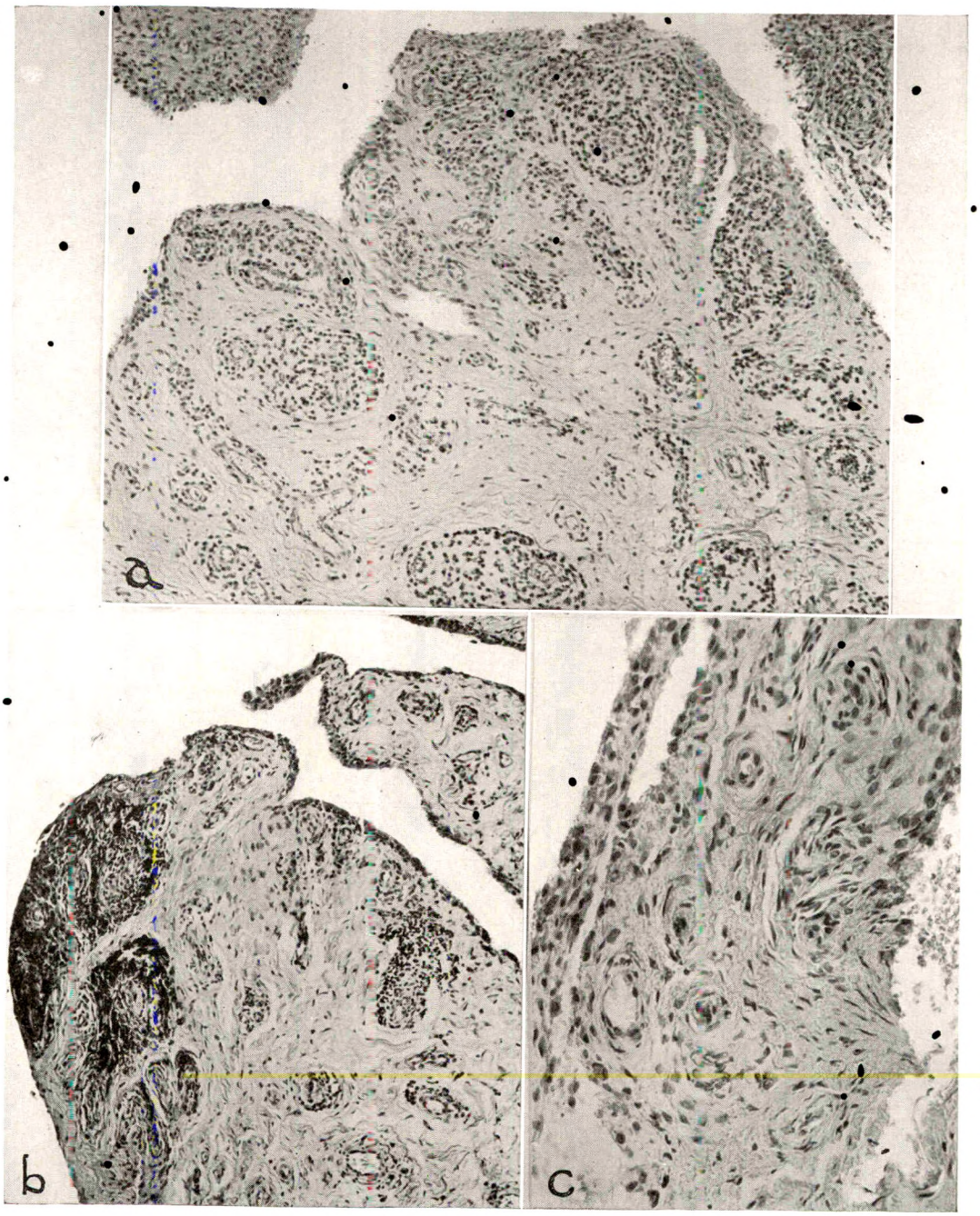


FIG. 2. *a*. Photomicrograph (×110) of synovia of right knee of patient W.M., *prior* to roentgen therapy. The surface lining cells appear normal. The subsynovial tissues are edematous, very vascular, and contain a diffuse sprinkling and many focal perivascular collections of chronic inflammatory cells.

b. Photomicrograph (×110) of synovia of left knee of the same patient, *following* a course of roentgen therapy (1,505 r to inner and 1,505 r to outer fields). The surface cells stain deeply but show no abnormal changes. The subsynovial tissues show a diffuse fibrosis, the edema has disappeared, the vessels are no longer dilated and are less numerous, and, in some instances, show obliteration of their lumina. There are scattered, small perivascular collections of lymphocytes, and to the left of the section, these contain many macrophages that are filled with blood pigment granules.

c. Photomicrograph (×220) of another area of the synovia of the irradiated left knee of W.M. This illustrates the marked fibroplasia in the subsynovial tissues, the thickening of the vessels walls, and the narrowing and obliteration of the lumina of some of the vascular channels.

number of treatment-series and interval (cumulative) periods.

Roentgen therapy was administered only to the inner aspect of the joint, because this side was more easily approached and because the small dimensions of the peripheral articulations in the dogs did not appear to require bilateral fields. Dogs No. 4, No. 1, No. 5 and No. 3 were killed after variable periods following the completion of roentgen therapy. The treated and control articulations were roentgenographed prior to and following the full course of therapy, and, at death, these joints were removed for histopathologic study. Dog No. 3 received the longest course of treatment (three and one-half months), the largest total dose to any one joint (8,000 r to the left knee joint), the largest combined total dose to all of the treated joints (8,000 r to the left knee joint, 4,000 r to the left elbow joint and 3,000 r to the right hip joint), and the longest cumulative periods. In order to evaluate "delayed roentgen-ray effect" and the effect of time more critically, dogs No. 2 and No. 6 are being observed for three and five years, respectively, following their courses of roentgen therapy.

Results of Roentgen Therapy in the Dogs.

Clinical. Only dog No. 1 showed loss of fur and some superficial skin ulceration over the left knee that had been subjected to superficial therapy. None of the dogs, including No. 1, evidenced any apparent discomfort in the treated joints up to their death. They manifested no limp and no apparent pain or tenderness; there was no swelling, and the passive and active ranges of motion remained unimpaired. This has also been true in the 2 living dogs which have been observed from the onset of their treatment on July 17 and August 4, 1941, to the present (February, 1942).

Roentgenographic. There were no changes in the roentgenograms of the treated and control joints made prior to treatment and just before death. Nor have there been any changes, thus far, in roentgenograms of the joints of the living dogs.

Pathologic. Gross examinations and histopathologic studies of the treated and control joints in dogs No. 4, No. 1, No. 5 and No. 3, including the articular cartilage, subchondral bone, synovial tissue, joint capsule and adjacent muscle tissue, revealed no abnormal changes in any of the specimens and no differences between the treated and control joints (Fig. 3, 4, 5, 6 and 7).

DISCUSSION

Roentgen therapy was utilized, with success, to control the effusions in a case of intermittent hydrarthrosis of the knee by May, Huet and Barnaud[20] (1937), and by Epstein and Edeiken[9] (1940). No recurrence was observed by the former authors in their case for ten months, nor by the latter authors in their case for twenty-six months. Intermittent hydrarthrosis is characterized by a periodically recurring effusion in the affected joint, of several months' duration, with intervals in which the joint is symptom free and functionally normal. The association of intermittent hydrarthrosis with rheumatoid arthritis has been observed by several authors (Krida[14]). In the experience of Ghormley and Cameron,[12] instances of "chronic synovitis" and of "intermittent hydrarthrosis" are in reality manifestations of chronic infectious arthritis, for in their cases other joints became affected subsequent to synovectomy. Cecil[7] has seen several cases of chronic intermittent hydrops develop typical rheumatoid arthritis. Inge[21] has also seen cases of chronic monarticular villous synovitis prove to be the precursors of generalized rheumatoid arthritis. For this reason no effort has been made to classify the joint involvement in our cases.

The use of roentgen rays in the treatment of the arthritides is not being evaluated in this study. Although recent, carefully controlled, clinical studies indicate that this form of treatment is of no value in rheumatoid arthritis involving the joints of the extremities (Smyth, Freyberg and Peck[24]), but is of value in the relief of symptoms in rheumatoid arthritis of the

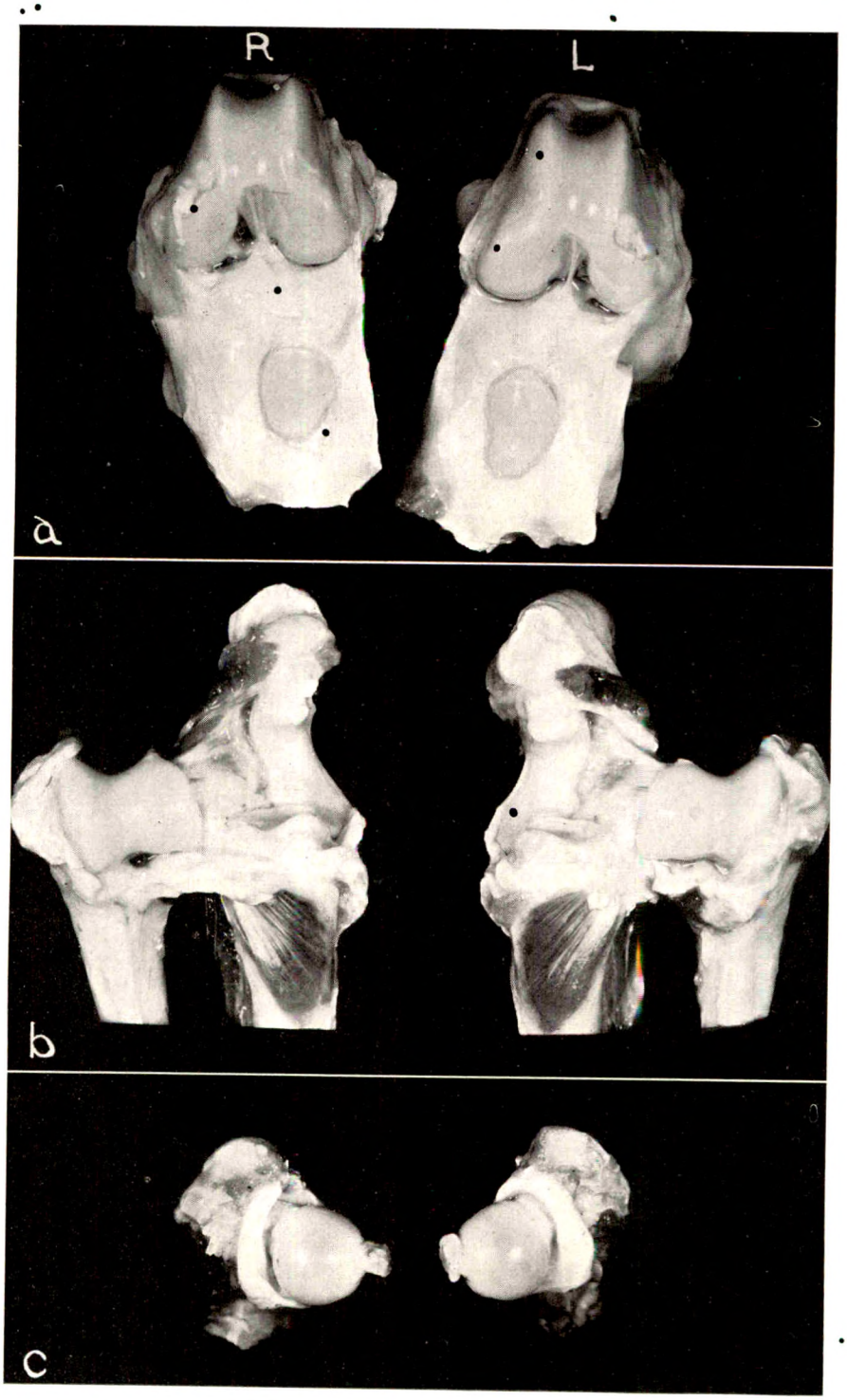


FIG. 3. Dog. No. 3. *a*, left knee joint (*L*) treated with 8,000 r (deep therapy) and untreated right knee joint (*R*). *b*, left elbow joint (*L*) treated with 4,000 r (deep therapy) and untreated right elbow joint (*R*). *c*, right hip joint (*R*) treated with 3,000 (deep therapy) and untreated left hip joint (*L*). There are no gross differences in the osseous and para-osseous tissues of the treated and control joints.

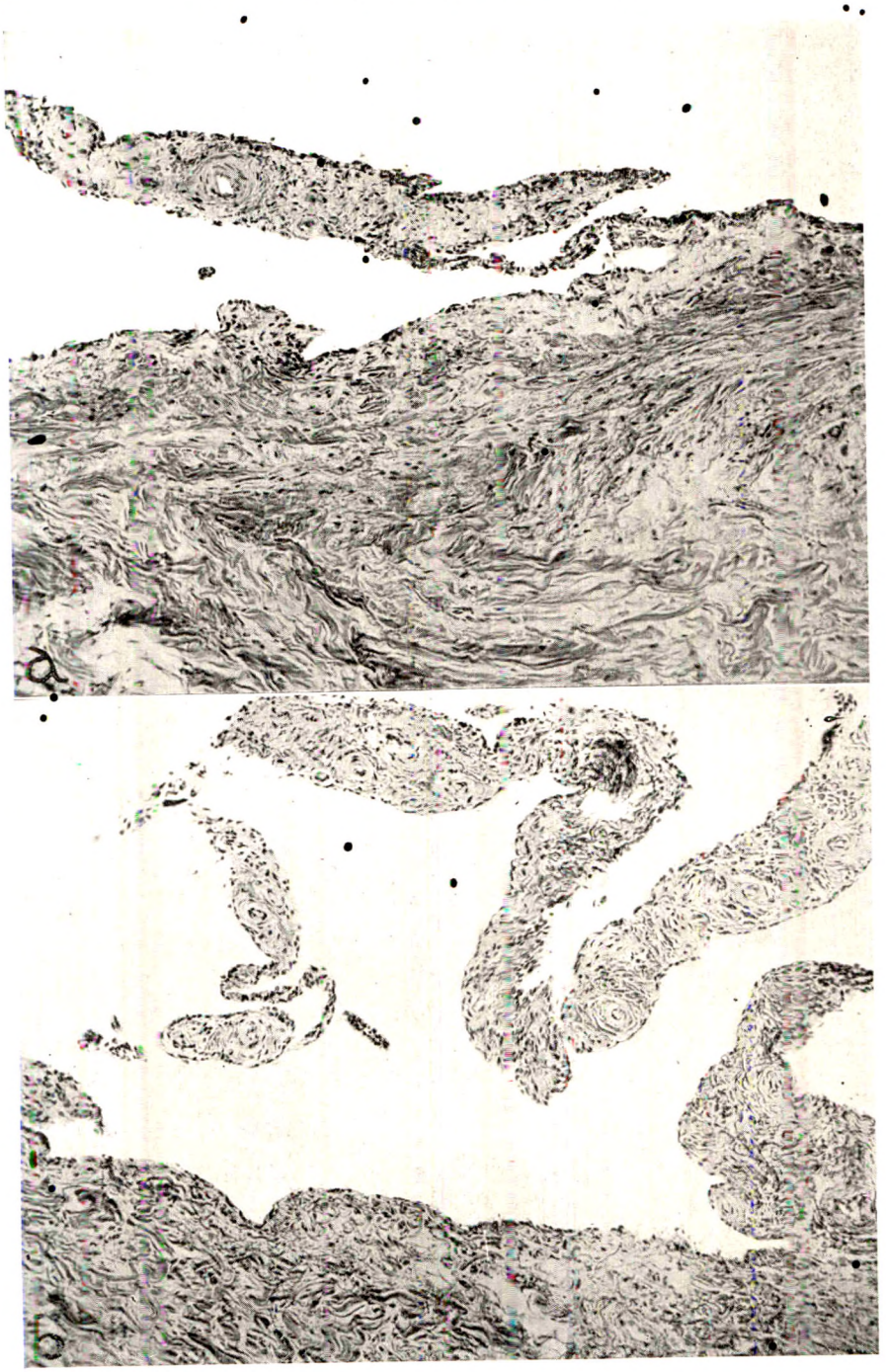


Fig. 4. Dog. No. 3. *a*, photomicrograph (×110) of the synovia of treated left knee joint (Fig. 3*a*). *b*, photomicrograph (×110) of the synovia of control (untreated) right knee joint (Fig. 3*a*). The histologic findings are normal and identical in both specimens.

spine (Smyth, Freyberg and Lampe[23]), no adequate explanation has been offered for the modus operandi in these cases.

Although the joint effusion and its attendant discomfort have been eliminated in the 8 affected knee joints in 5 of our 6 cases, there is no reason to suggest that the roentgen therapy in these cases will also

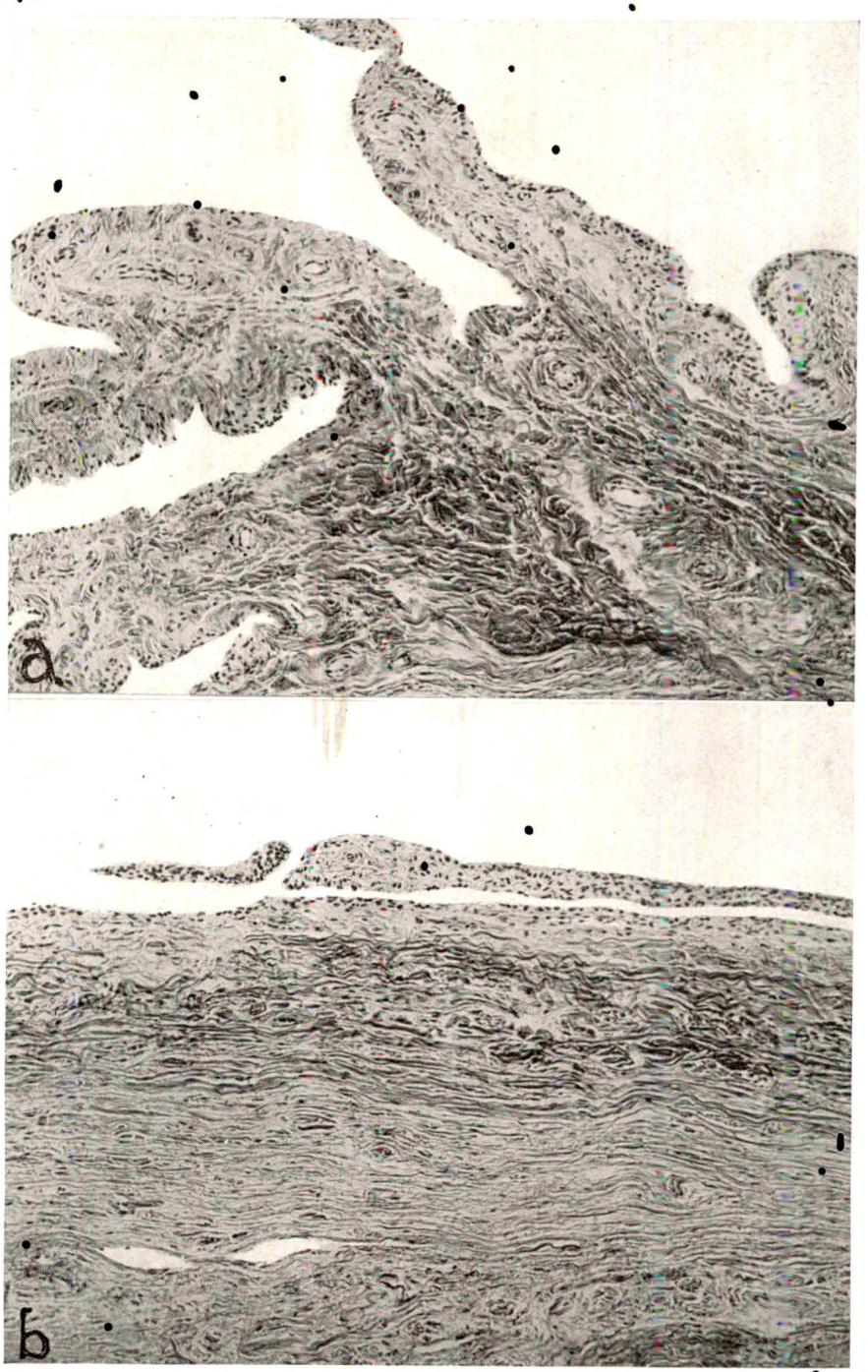


FIG. 5. Dog. No. 3. *a*, photomicrograph (×110) of the synovia of treated left elbow joint (Fig. 3*b*). *b*, photomicrograph (×110) of the synovia of control (untreated) right elbow joint (Fig. 3*b*). The histologic findings are normal and identical in both specimens.

serve to favorably affect the progress and ultimate course of the underlying disease processes. Indeed, the residual asymptomatic synovial thickening in the treated joints of M.D. and F.S., and the painful synovial thickening in the irradiated knee



Fig. 6. Dog. No. 5. *a*, left knee joint (*L*) treated with 4,000 r (deep therapy) and untreated right knee joint (*R*). *b*, left elbow joint (*L*) treated with 2,000 r (deep therapy) and untreated right elbow joint (*R*). There are no gross differences in the osseous and para-osseous tissues of the treated and control joints.



FIG. 7. Dog. No. 4. *a*, left knee joint (*L*) treated with 3,000 r (superficial therapy) and untreated right knee joint (*R*). *b*, left elbow joint (*L*) treated with 3,000 r (deep therapy) and untreated right elbow joint (*R*). There are no gross differences in the osseous and para-osseous tissues of the treated and control joints.

joint of S.G., indicate the persistence of the pathologic joint lesions in these cases. We believe that certain features in the pathologic specimens removed from 2 cases following roentgen therapy may represent some of the responsible factors in the con-

trol of the joint effusions by this form of treatment. These features are: (1) the disappearance of edema, (2) the increase in fibrosis and (3) the thickening of the vessel walls and obliteration of the smaller vascular channels in the subsynovial tissues.

The great sensitivity of vascular endothelium is one of the most important factors in radiation therapy. The mode of action of radiation on tumors, for example, is generally believed to be due partly to the direct action on a certain proportion of the tumor cells, and partly to the damaging effect on the circulation (Pohle,[21] Warren[27]). While lymphoid cells, polymorphonuclear leukocytes, and epithelial cells are very sensitive, connective tissue, muscle, cartilage and bone tolerate large exposures to radiation (Desjardins[8]), except such as may be interpreted as interfering with their blood supply. Changes in the lymph flow probably play a similar rôle but are difficult to demonstrate (Ewing[10]). In bone, such vascular changes may cause late bone necrosis, appearing months after treatment has ceased (Ewing,[11] Watson and Scarborough[28]). The neck of the femur has been found to be particularly vulnerable to the vascular changes incidental to roentgen therapy for pelvic malignancy (Strauss and McGoldrick[26] and others[1,3,13]).

The experiments of Lacassagne and Vinzent[19] and Lacassagne[16,17,18] demonstrated the occurrence of rabbit sarcoma in regions of acute inflammation, induced by injecting *Streptobacillus caviae* and exposed to irradiation by roentgen rays. There have been a number of clinical reports of sarcoma following roentgen therapy of human joints, the pathologic lesion prior to treatment being definitely tuberculous in some of the cases (Küttner,[15] Decker[4]). Because the malignant changes did not become manifest in these reported cases for several years, our clinical material, as well as the living dogs No. 2 and 6, will be observed for three to five years following treatment.

The total dosage in our human cases has been kept low, and has been fractionated. Despite the experience that much larger doses applied to the more vulnerable joints of our dogs were without immediate and, thus far, remote deleterious effects, it is our experience that very heavy roentgen-ray dosage will affect normal human joints adversely. Painful and disabling swelling, loss of motion and roentgenographic evidence of a destructive arthritis have been observed in the normal knee joint adjacent to a heavily irradiated benign osseous lesion involving the medial femoral condyle in a twenty-nine year old female, and the upper tibia in a fifteen year old boy. In a third case, a seventeen year old male with a benign osseous lesion in the lower end of the femur that had been heavily irradiated, the adjacent, partially ankylosed knee joint was examined histopathologically following its excision for the correction of a flexion deformity.* It was observed that the synovia had suffered little or no change. There was increased vascularity and a diffuse sprinkling of chronic inflammatory cells in the subsynovial tissues, with a tendency to the formation of focal perivascular collections. The articular cartilage had suffered considerable change—in some areas being thinned or absent, in others being frayed, or calcified, or covered by granulation tissue (pannus). The subchondral bone was very porotic, with increased marrow fibrosis. Despite all of these degenerative manifestations there was no evidence of malignant transformation. The benign tumor tissue in this specimen was well removed from the articular tissues and it was interpreted that these articular changes had followed the intensive roentgen therapy to the juxta-articular tissues.

Bisgard and Hunt[5] and others[6,22] have observed retardation of bone growth in young laboratory animals following irradiation of the epiphyseal plates. Barr, Lingley and Gall[2] observed that within a dosage of 1,000 to 1,335 r (200 kv.) sufficient damage occurred to the epiphyseal plate to cause

* Histologic specimens in this case were examined in the Laboratories of Pathology of the Hospital for Joint Diseases, New York City; Director, Dr. Henry L. Jaffe.

growth retardation of about 50 per cent in laboratory animals, but that within this range the effects noted in the articular cartilage, synovia, skin and muscle were negligible, and the bone marrow changes appreciable but localized. These experiments have been extended as a therapeutic measure by Spangler[25] to secure epiphyseal growth arrest in 3 children and he has concluded that around 3,000 r, given in one fractionated series, will produce epiphyseal closure in humans. This author observed no clinical evidence of soft tissue change or damage, and no roentgenographic evidence of alteration in bone trabeculation in the irradiated areas as late as nine months after treatment. These observations lend support to our present belief that roentgen therapy, in the doses administered by us, is without deleterious effect to human knee joints that are the site of nonspecific synovial lesions and to the normal joints of the extremities of dogs.

CONCLUSIONS

Roentgen therapy has been found beneficial in the *control of the joint effusion* and its attendant discomfort in certain nonspecific synovial lesions involving the knee joint in humans. Within the recommended doses, no immediate and, as yet, no remote deleterious effects have been observed in these cases. The effusion was eliminated in eight knee joints in five patients, and no change was effected on the synovial effusion of the knee joint in the sixth case following an incomplete course of roentgen therapy. Residual asymptomatic synovial thickening has persisted in three knee joints of two patients and painful synovial thickening in the knee joint of one patient. There is, therefore, nothing to suggest that roentgen therapy has served or will serve to affect favorably the progress and ultimate course of the underlying pathologic processes in these cases.

The more vulnerable articulations of the extremities of six normal adult dogs, treated with much larger doses than have been utilized and found effectual in the human subjects, have shown no immediate, and, thus far, no remote untoward effects.

The effect of such factors as type, dose and duration of roentgen treatment have been analyzed in this preliminary report. "Delayed roentgen-ray effect" and the time factor must be evaluated in the human subjects and in the two living dogs, and these findings will be presented in a subsequent report.

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STEREOSCOPIC DEPTH PERCEPTION*

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WHILE TIME and thought have been spent on the theory of stereoscopic roentgenography and upon the development of technical apparatus, unfortunately little attention has been given to the personal ocular equipment of the radiologist. The state of the radiologist's own ocular and oculomotor apparatus, and the degree of his ability to fuse stereoscopic images into a satisfactory three-dimensional whole are commonly unknown quantities. Arguments over interpretation of stereoroentgenograms might disappear if all persons viewing them had the same development of stereoscopic vision. One of us (Batson) has been interested for a long time in the way in which physicians and medical students respond to the roentgen stereoscope. Some staunchly maintain that the three-dimensional image can be made to appear as if viewed from the posterior or the anterior aspect at will. This ability to interchange the appearance of the image disappeared in three persons after their vision was corrected. With such experiences in mind it was thought advisable to investigate the exact state of the stereoscopic vision of our trainees in roentgenology in the Graduate School of Medicine. The cooperation and approval of the Vice Dean for Roentgenology, Dr. George E. Pfahler, and of the then Vice Dean for Ophthalmology, the late Dr. William T. Shoemaker, were readily obtained. The first group of student-physicians was examined in the academic year 1941–1942, in the Orthoptic Clinic of the Graduate Hospital, under the direction of the junior author. Examinations were carried out again this year (1943) on the group at present enrolled.

It is obvious that a physician with little or no depth perception can obtain slight benefit from stereoscopic roentgenograms. Such a one should be cognizant of his own deficiency, and might possibly limit his examinations to "flat" roentgenograms. Further, a defective stereoscopic sense might be improved by the use of orthoptic exercises or correction of any existing refractive error.

Three groups of tests were used. First, a check was made of the visual acuity and muscle balance. The visual acuity of the physician was determined both with and without his usual optic correction, if any. One of these examined was wearing a wholly inadequate correction. He realized that he should consult his ophthalmologist, but he had no idea that he was depending largely upon one eye.

The group was next tested for the ability to fuse the right and left eye images into a single whole. All of those tested had this ability; in fact, all had what is technically known as "gross third degree fusion." That is to say, all could see a pair of stereoscopic pictures of a box or a barrel as a three-dimensional object. This test is carried out with suitable cards, and a Brewster type stereoscope. Since all of our group had two functioning eyes, it might be assumed that long before entering medical school fusion of this degree would have been developed. Roentgenology is probably not a specialty favored by anyone with the handicap of only one functioning eye.

It should be emphasized that all of those examined were familiar with the roentgen stereoscope, and had had some experience in its use. All thought that they were able to use the stereoscope satisfactorily, because they could see a chest "in the round." This ability would be expected since all had "gross third degree fusion." Some knew that they could not see all of the details in stereoroentgenograms that would be de-

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scribed by others, but did not believe that this was due to any deficiency in their personal visual equipment.

The third group of tests was the most significant since it determined the amount of critical depth perception, known technically as stereopsis. This is the faculty necessary to perceive depth relationships—important in accurate roentgen interpretation. The degree of stereopsis was determined with suitable image cards, and a hand stereoscope. In these tests the Keystone stereometric cards were used, and the lower the percentage, the poorer the stereopsis. Seventy per cent of stereopsis is considered the low limit of normal, while provision is made for ratings as high as 110 per cent. Not knowing the minimum percentage required in roentgenologic interpretation, we decided to accept 70 per cent as normal.

Binocular vision is the ability to use both eyes simultaneously to produce a single mental impression. In "third degree fusion" this faculty is developed to the extent that the slightly dissimilar and separate images, as perceived by the two eyes, can be blended into one which has a sense of depth or perspective. Fusion is a cerebral function and is first manifest about six to eight months after birth. This depth sense is developed through use. The more this fusional ability is used, the more perfect will fusion become. It is not clear how late in life one may increase the fusional ability, hence we note the ages of those examined.

Imperfect balance of the extraocular muscles, that is a tendency of the visual axis of each eye to deviate from that of the other, will naturally impair fusion and stereoscopic perception. Also, errors of refraction contribute to difficulty in fusion in several ways. If one eye is much more nearsighted or farsighted than the other, or if one eye is nearsighted and the other farsighted, the images on the two retinae will be of different sizes and degrees of sharpness. These images will therefore be difficult to fuse. Uncorrected astigmatic errors cause distortion, and hence dissimilarity of the two retinal images. The farsighted have blurred images at the near point. The nearsighted individual may find that since he does not have to use his accommodation fully at the near point, the convergence-accommodation ratio is so disturbed that fusion becomes difficult. When the age is reached that the observer depends upon bifocal glasses to supplement his accommodation, he must be trained to use his convergence to the same degree as formerly. Proficiency in depth perception decreases if this is not done.

Another cause for a low percentage of stereopsis in an otherwise normal individual is lack of attention. This individual will have to learn to use this power of observation.

In all, 16 physicians were examined. Ten had average, or better than average, depth perception. The ages of the 10 ranged from twenty-six to thirty-nine years; the percentage of stereopsis from 70 to 107 per cent. There was no age correlation. At the time of examination, no uncorrected refractive errors were found in this normal group. None needed exercises for muscle imbalance.

The other 6 physicians presented enough individually interesting features to warrant a brief summary of each case.

Case I, aged twenty-nine, had one degree of stereopsis. By correction of his muscle balance, with the practice use of prisms, this doctor was able to bring his stereopsis up to 30 per cent. His problem was discussed with him, and he was advised to continue the investigation further, when impending military duty permitted. Even 30 per cent stereopsis should be of an advantage.

Case VIII, aged twenty-seven, had a stereopsis of 35 per cent. After a two hour period of explanation, and experimentation with fusion cards, he brought his perception up to 88 per cent. In other words, he had normal ocular and oculomotor equipment, but had never learned to use it in the manner required in the test. He may have no further difficulty now that he is conscious of the problem.

Case X, aged thirty-two, is one of the most interesting. When examined in January, he

had a stereopsis of 45 per cent. He began the daily use of exercise cards, but it was not until July that he brought his stereopsis up to 90 per cent. He believed that his ability to interpret stereoscopic roentgenograms increased concurrently. Unfortunately, the significance of the test percentages in terms of roentgen interpretation is not known. It may be necessary for this physician to continue the use of the exercise cards to maintain his improvement.

CASE XII, aged forty, when first examined did not have a proper correction of his refractive error. His stereopsis was 25 per cent. After refraction his stereopsis still remained at 25 per cent. Following his second examination, he used the exercise cards proper for his type of muscle imbalance; when again retested ten days later his depth perception was 100 per cent

CASE XIII, aged thirty, had 10 per cent stereopsis, but following a thirty minute period of explanation and experimentation, this became 90 per cent.

CASE XIV, aged thirty-nine, stereopsis 28 per cent, was, like the preceding case, able to bring this to 97 per cent after a one-half hour instruction period.

To summarize, 10 out of a group of 16 beginners in roentgenology had adequate depth perception. Three more with normal oculomotor and ocular apparatus had not come to appreciate the significance of seeing stereoscopically, at least not to the degree required in the test used. A short practice period increased their test percentage to normal range. The remaining 3 did not improve with a short practice period. One of these required ten days, one six months. The remaining case, on whom we were unable to complete our studies, had a defect in muscle balance which could be partially corrected by the practice use of prism exercises. This man expects to follow through at a later date.

It is a pleasure to acknowledge the interest and the close cooperation of the entire group undergoing these tests. Although the correlation between test and roentgen practice is not known, from the diagnostic and remedial results obtained with this small group, we venture to suggest that all beginners in radiology have the exact state of their stereoscopic vision determined. The daily use of the roentgen stereoscope is in itself good stereoscopic training, yet established roentgenologists might well avail themselves of an examination of their stereoscopic efficiency. This examination would be especially revealing at the age period when near vision begins to recede.

amine the patient in the lateral upright position, when the Meckel's diverticulum would be seen anteriorly. The length of time the diverticulum remains visible depends on the size of the pouch, its position and the width of its neck.

CONCLUSION

A classical roentgen demonstration of a Meckel's diverticulum is presented.

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VOL. 51, NO. 2

THE ROENTGEN DEMONSTRATION OF MECKEL'S DIVERTICULUM*

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ALTHOUGH numerous and adequate descriptions of Meckel's diverticulum have appeared in the literature, the illustrations accompanying these papers usually fail to demonstrate the pouch very clearly.

This case is presented because it represents a classical visualization of a Meckel's diverticulum. Fortunately, the entire diverticulum, its neck and the ileum proximal and distal to the diverticulum, are completely visualized, enabling one to gauge the size of the pouch, its shape, position, relation to the small bowel, and its distance from the ileocecal valve.

The omphalomesenteric or vitelline duct (the duct of communication between the yolk sac and the primitive digestive tube in early fetal life) normally disappears within a few weeks after birth, but it occasionally persists more or less as a fibrous cord, a tube or a diverticulum. In the latter instance, the diverticulum is due to the persistent patency of the proximal or mesenteric end of the duct. Anatomically, it is found in about 1 to 3 per cent of individuals and is usually situated 1½ to 3 feet above the ileocecal region in the cadaver. It springs from the antimesenteric border, and its blind end is usually free or it may be connected to the abdominal wall or some other portion of the bowel. Usually, the diverticulum has the same morphology as the ileum, but occasionally aberrant tissue, such as gastric mucosa or pancreatic tissue, has been found in the diverticulum. In the former instance peptic ulcers have been found. Roentgenographically, the visualization of a Meckel's diverticulum is a rather infrequent occurrence.

CASE REPORT

The patient, a woman aged sixty, had had four previous operations, the first at the age of twenty for appendicitis, followed by operations for intestinal adhesions, ventral hernia and, finally, a hysterectomy.

Barium enema examination revealed an ileal diverticulum 18 inches from the ileocecal valve. It was 2.5 cm. long and 2 cm. at its widest diameter. It was symmetrical and pear shaped, with smooth borders, connected with the ileum by a neck 1.5 cm. long and 1 cm. wide (Fig. 1). Roentgenoscopically, its blind end was free and not tender. The diverticulum was removed and was found to have the same morphology as ileum. No aberrant tissue was found.

Roentgenographically, Meckel's diverticulum may be visualized by means of the oral fractional method or by barium enema. An additional roentgen method is to ex-

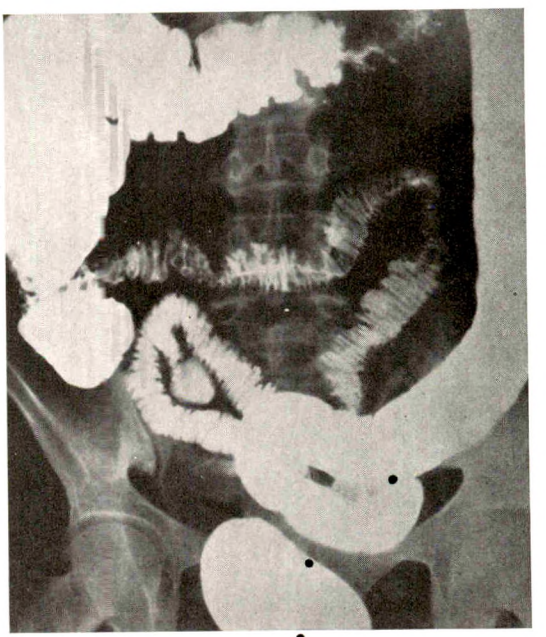


FIG. 1. The Meckel's diverticulum is seen in the right lower quadrant arising from the loop of ileum which is about 18 inches from the ileocecal valve on the roentgenogram.

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A PRACTICAL TECHNIQUE FOR ROENTGEN PELVIMETRY WITH A NEW POSITIONING*

By A. E. COLCHER, M.D., *and* WALTER SUSSMAN, M.D.
PHILADELPHIA, PENNSYLVANIA

NUMEROUS techniques such as those by Jarcho, Thoms, Caldwell and Moloy, Snow, Weitzner, and others have been described. Each has given good results, but no one method has been found to be universally adopted nor embodies the following prerequisites:

1. Simple intersecting diameters of the true pelvis.
2. Simple positioning of the patient.
3. A simple ruler for rapid and direct mensurations comprehensible to the roentgenologist, obstetrician and general practitioner.

A method which fulfills these basic requirements is described in this paper.

I. INTERSECTING DIAMETERS OF THE PELVIS

We have divided the true pelvis into three levels with intersecting anteroposterior and lateral diameters. These levels include all the salient bony landmarks of the pelvis (Fig. 1, *C–D*).

Actual Inlet (First Level)

Lateral View. The actual inlet, as emphasized by us, starts from the inner upper margin of the symphysis pubis and extends along the iliopectineal lines through the level of the transverse diameter of the inlet and is projected to the sacrum (Fig. 2). The anteroposterior diameter of this strait is the line from *G* on the upper margin of the symphysis pubis to point *I* on the sacrum. Accordingly, on the lateral view, line *G–I*, is drawn from the upper inner symphysis through the point *E* to the sacrum *I*. Point *E* is midway between the brim of the pelvis above, and the sciatic notches below; and about one-third the distance from *I* to *G*.

This we consider as the anteroposterior diameter of the actual inlet.

Anteroposterior View. The transverse diameter line is drawn at the widest points of the inlet *A–A′*.

In this manner, the anteroposterior diameter *G–I* intersects the transverse diameter *A–A′* on the same level at point *E*.

Mid-Pelvis (Second Level)

Lateral View. The mid-pelvis anteroposterior diameter is developed from the inner lower border of the symphysis, *P*, and projected through the mid-point between the spines of the ischium, *F*, to the inner aspect of the sacrum, *M* (Thoms[4]).

Anteroposterior View. The transverse diameter is measured between the spines of the ischium *B–B′*.

The above diameters intersect at point *F* and form the mid-pelvis diameters.

Outlet (Third Level)

Lateral View. The anteroposterior diameter is a line drawn from the tip of the sacrum *S* to the mid-tuberal diameter *T*. The bituberal points in this view are obtained by projecting straight lines from the lower border of the obturator foramen to the edge of the tuberosities of the ischium, *C–C′* (Snow[2]).

Anteroposterior View. The transverse diameter of the outlet is obtained by drawing a line from the lateral margin of the inlet, projected along the lateral wall of the forepelvis (noted as a dense white line on the roentgenogram) to the lower edge of the tuberosity of the ischium. With the same procedure on both sides, the transverse diameter *C–C′* is obtained (Snow[2]).

Thus, the anteroposterior diameter *S–T* intersects the transverse diameter *C–C′* at

* From the Departments of Radiology and Gynecology, Doctors Hospital, Philadelphia. Presented before the Philadelphia Roentgen Ray Society, May 6, 1943.

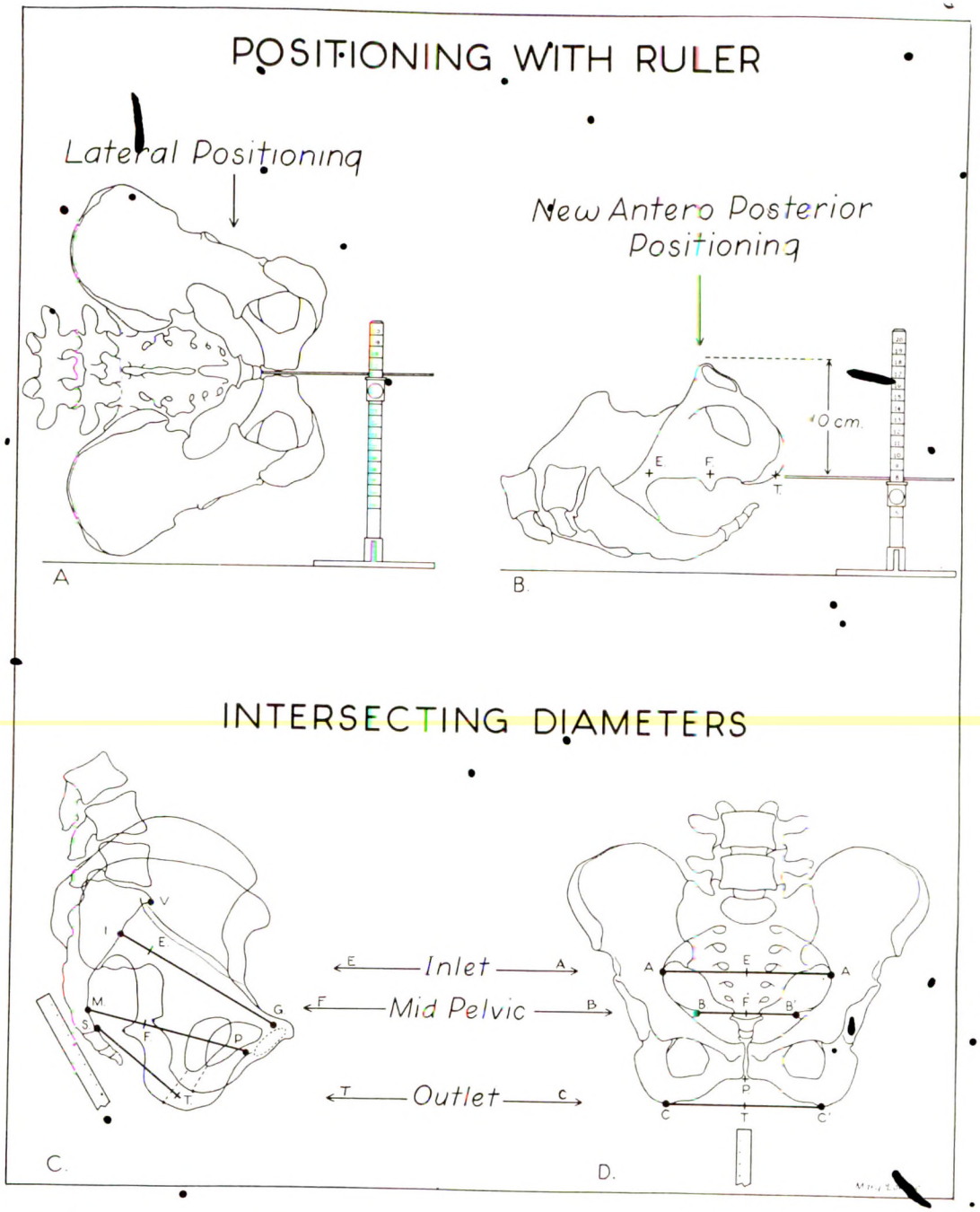


FIG. 1. *A* and *B*, diagrams showing lateral and anteroposterior positions with ruler. *C* and *D*, diagrams of the three pelvic levels with intersecting diameters.
Inlet: Actual anteroposterior diameter of inlet line *G–I* crosses through inlet transverse diameter *A–A'*.
Midpelvis: Anteroposterior diameter *P–M* crosses transverse diameter *B–B'*.
Outlet: Anteroposterior diameter of outlet *T–S* bisects midpoint of transverse diameter line *C–C'*.

the mid-tuberal point *T*.

True Conjugate. This is measured on the lateral view from a point between the upper inner aspect of the symphysis to the sacral promontory, *G–V*. It will be noted that there is no measurable transverse diameter on this level that would be of any value, since it would project into the false pelvis.

Therefore, the true conjugate is measured and considered as a separate entity.

Fetal Head

The ideal time for roentgen examination is at the beginning of labor or one week prior to the expected date of birth. The fetal head should be in the midline, otherwise fetal measurements are not considered accurate. We have taken two measurements of the fetal skull, the smallest and the greatest diameters in both the anteroposterior and lateral views. The average is recorded.

2. POSITIONING

Lateral View (Fig. 3*A*). This is the generally used true lateral exposure centered over the great trochanter. This results in a sagittal cross section through all the anteroposterior diameters of the pelvis. The fetal head, if in the midline, can also be measured (Granzow,[4] Weitzner,[6] Thoms[5]).

New Positioning for Anteroposterior View

Anteroposterior View (Fig. 3*B*). Since the lateral view just described has been conceded to be accurate, it now logically follows that any positioning in the anteroposterior exposure that would bring all the salient transverse diameters on the same level would result in an ideal setup for direct mensuration.

To meet this requirement, we have placed the patient flat on the back with the knees semiflexed and separated. This elevates the forepelvis and brings the bituberal diameter, bi-ischial diameter and the transverse diameter of the inlet practically on the same level and in the same plane as the table and film. Figure 4 shows a roentgenogram with diameters in lateral view. Figure 5 shows a roentgenogram with diameters in anteroposterior view.

3. ROTATING RULER*

A ruler devised by us (Fig. 1, *A* and *B*) consisting of an opaque metal strip with true centimeter scale perforations is used. This ruler is swiveled on a projecting arm

* May be obtained from Picker X-Ray Corporation.

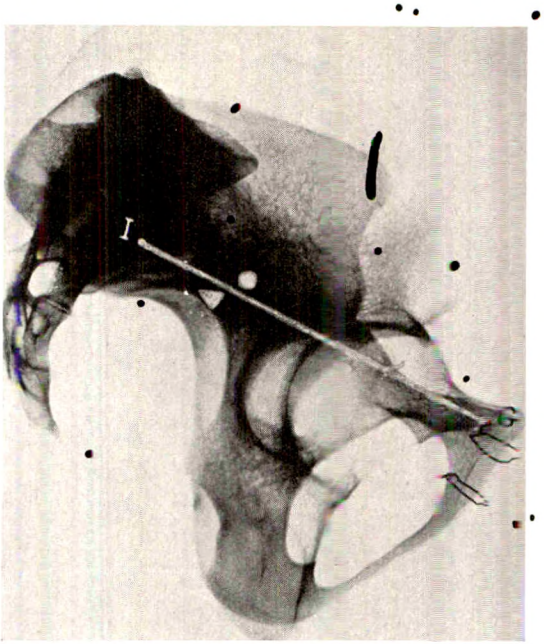


FIG. 2. Lateral roentgenogram of dried pelvis. Line *G–I* is actual anteroposterior diameter of inlet passing through the middle of the transverse diameter of the inlet as shown by lead markers.

attached to a vertical stem, which is also calibrated in centimeters. The stem is attached to a base parallel to the perforated scale. The ruler may be elevated, lowered and rotated. *Regardless of position, the scale is always parallel with the table top and the film.* Thus, when roentgenographed with the patient, the ruler will have the same distortion as the diameters on the same level. Therefore, the ruler markings on the film become the centimeter scale. These markings can be transferred to a sheet of paper, or a special chart to be described later, and used for the necessary measurements.

Placement of Ruler

Lateral View (Fig. 1*A*). The ruler is placed at the mid-sacral spine parallel with the spine and film. Thus the centimeter rule markings are projected on the film for direct mensuration. It is simple and easy to accomplish.

Anteroposterior View (Fig. 1*B*). The ruler is placed at the level of the tuberosities of the ischium by direct manual palpation or

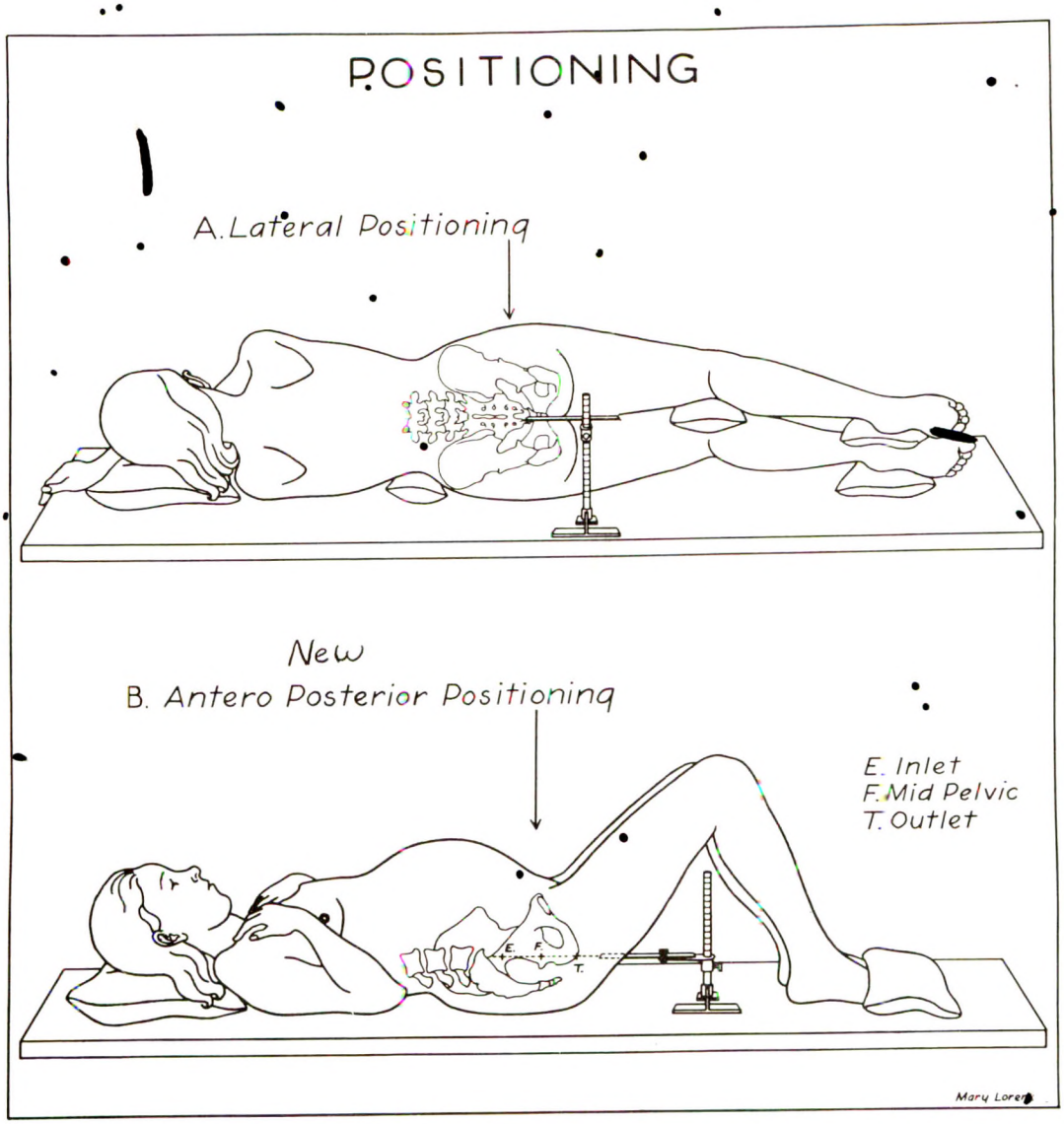


FIG. 3. *A*, lateral positioning. Patient on side; knee and thighs semiflexed. Ruler is at level of midsacral spine; the central ray is directed through the great trochanter of the femur any distance. *B*, new antero-posterior positioning. Patient flat on back; knees and thighs semiflexed and slightly separated. Ruler is placed at level with tuberosities of ischium. Central ray above symphysis at any distance.

lowering the ruler 10 cm. below the superior border of the symphysis pubis. The centimeter markings are projected on the film and have the same distortion as the transverse diameters of the pelvis.

The placement of the ruler 10 cm. below the top of the symphysis pubis is based on a study of 13 dried female pelves at the Wister Institute, 2 of our own, and living subjects. For soft tissues overlying the symphysis, 5 mm. were added. We have also dropped this ruler 8 and 12 cm. below the top of the symphysis pubis and the readings of the specimens did not vary more than 3 mm. to every 10 cm., or approximately 3 per cent variation. This includes the interspinous diameter which lies sometimes as much as 1.5 cm. below the level of the inlet and outlet.

Due to the proximity of the ruler to the table in this new position (about 4 inches) and with a 36 or 40 inch target distance, a slight

variation of the level of the transverse diameters of the pelvis becomes negligible in the final analysis.

The Chart (Fig. 6). It will be noted that the words lateral and anteroposterior are seen on the right and left margins of the chart. When the roentgenograms are ready, the chart edge marked lateral is placed on the ruler markings of the lateral view and the scale is marked off. A similar procedure is carried out with the anteroposterior view. The respective newly made rulers on the edges of the chart are used to measure the actual diameters on the roentgenograms.

EVALUATION OF DIAMETERS

The intersecting diameters of the actual inlet *G–I* and *A–A'* are added together. The total of 22 to 24 cm. is considered a normal range. The total of the mid-pelvis *M–P* and *B–B'* should average between 20 and 22 cm. Then the outlet *S–T* and *C–C'* are added; the normal range should average between 16 and 18.5 cm. (Snow[3]).

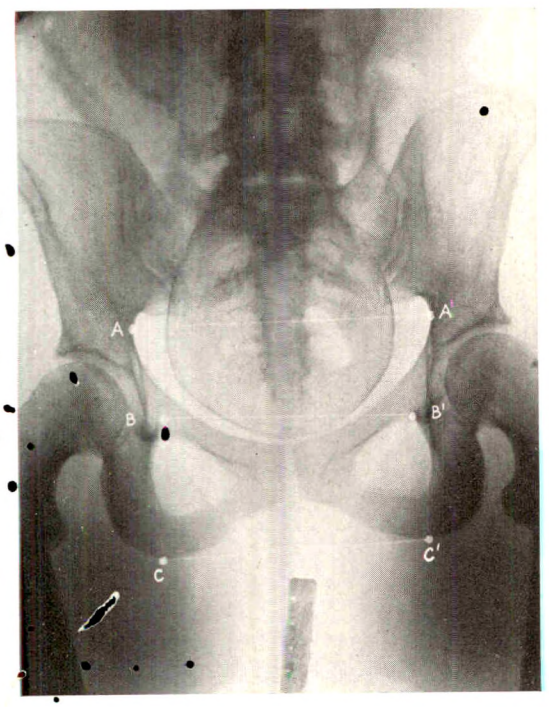


FIG. 4. Anteroposterior roentgenogram (as in diagram of Fig. 1*D*). *A–A'*, transverse diameter of inlet; *B–B'*, transverse diameter of mid-pelvis at the level of the ischial spines; *C–C'*, transverse diameter of outlet at level of tuberosities of ischium. Centimeter ruler markings on roentgenogram used for measurements.

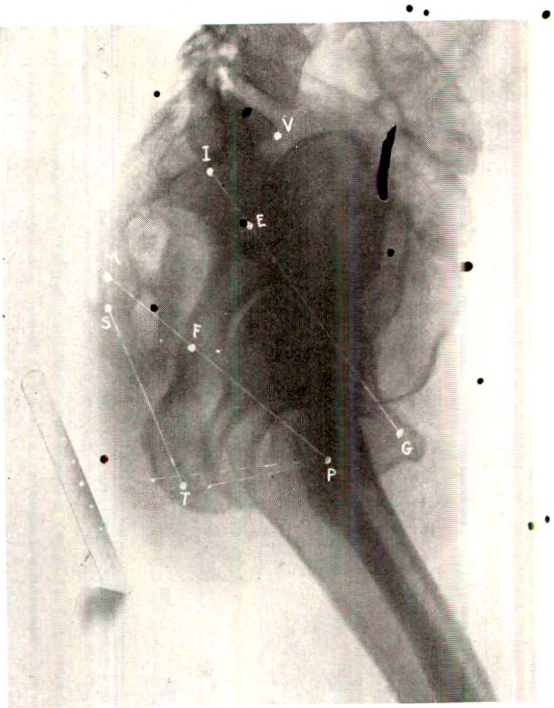


FIG. 5. Lateral roentgenogram (as in diagram of Fig. 1*C*). Line *G–V* is textbook true conjugate; *G–I*, anteroposterior diameter of actual inlet; *P–M*, anteroposterior diameter of mid-pelvis; *T–S*, anteroposterior diameter of outlet. Centimeter ruler markings on roentgenogram used for measurements.

The textbook true conjugate is evaluated on its own measurements as a separate diameter for obstetrical consideration. The normal range is 11 to 11.5 cm.

The fetal head measurements can be evaluated by the general average in both the anteroposterior and lateral views.

Consideration of the different pelvic diameters with the various measurements of the fetal head, such as the biparietal and the suboccipitobregmatic diameters, should also receive separate obstetrical evaluation. The latter diameters are easily measured when the fetal head lies in the anteroposterior or transverse position. The fetal head must be in the midline for accuracy.

SUMMARY

1. A simple positioning of the patient for roentgen pelvimetry has been described.

ANTEROPOSTERIOR VIEW

Centimeter Scale

Taken From

Film

CHART

TRUE CONJUGATE G - V

FETAL HEAD — Anteroposterior View; Lateral View; Average

		Diameters	TOTAL	NORMAL RANGE OF TOTAL
ACTUAL INLET	Anteroposterior	G - I		22 - 24
	Transverse	A - A'		
MID-PELVIS	Anteroposterior	P - M		20 - 22
	Bispinous	B - B'		
OUTLET	Anteroposterior	T - S		16 - 18.5
	Bituberal	C - C'		

Type of Pelvis — Lumbo-sacral articulation

Position of Fetal Head — Separation of Symphysis

Moulding of Fetal Head — Position of Coccyx

Remarks:

LATERAL VIEW

Centimeter Scale

Taken From

Film

FIG. 6. Sample chart. Ruler markings at sides taken from roentgenograms used as final ruler for measurements and records of pelvic diameters and fetal skull.

2. Only two exposures are required, an anteroposterior and a lateral view, taken at any distance.

3. The ruler gives direct centimeter measurements on each film without need of correction tables.

4. The true conjugate is considered as a separate entity since it has no transverse diameter in the true pelvis on the same plane.

5. The diameters of the pelvis—namely, those of the inlet, the mid-pelvis and the outlet—are measured along intersecting lines embracing all bony landmarks.

6. The fetal head, when presenting in the midline, can be measured in both the lateral and anteroposterior views.

7. A quick and accurate method for measuring the subpubic angle by measure of a precalculated table (Fig. 7) is included.

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DISCUSSION

DR. PAUL C. SWENSON, Philadelphia. Dr. Colcher and Dr. Sussman have added another relatively simple, easy method of pevimetry to the growing list of variations of one method

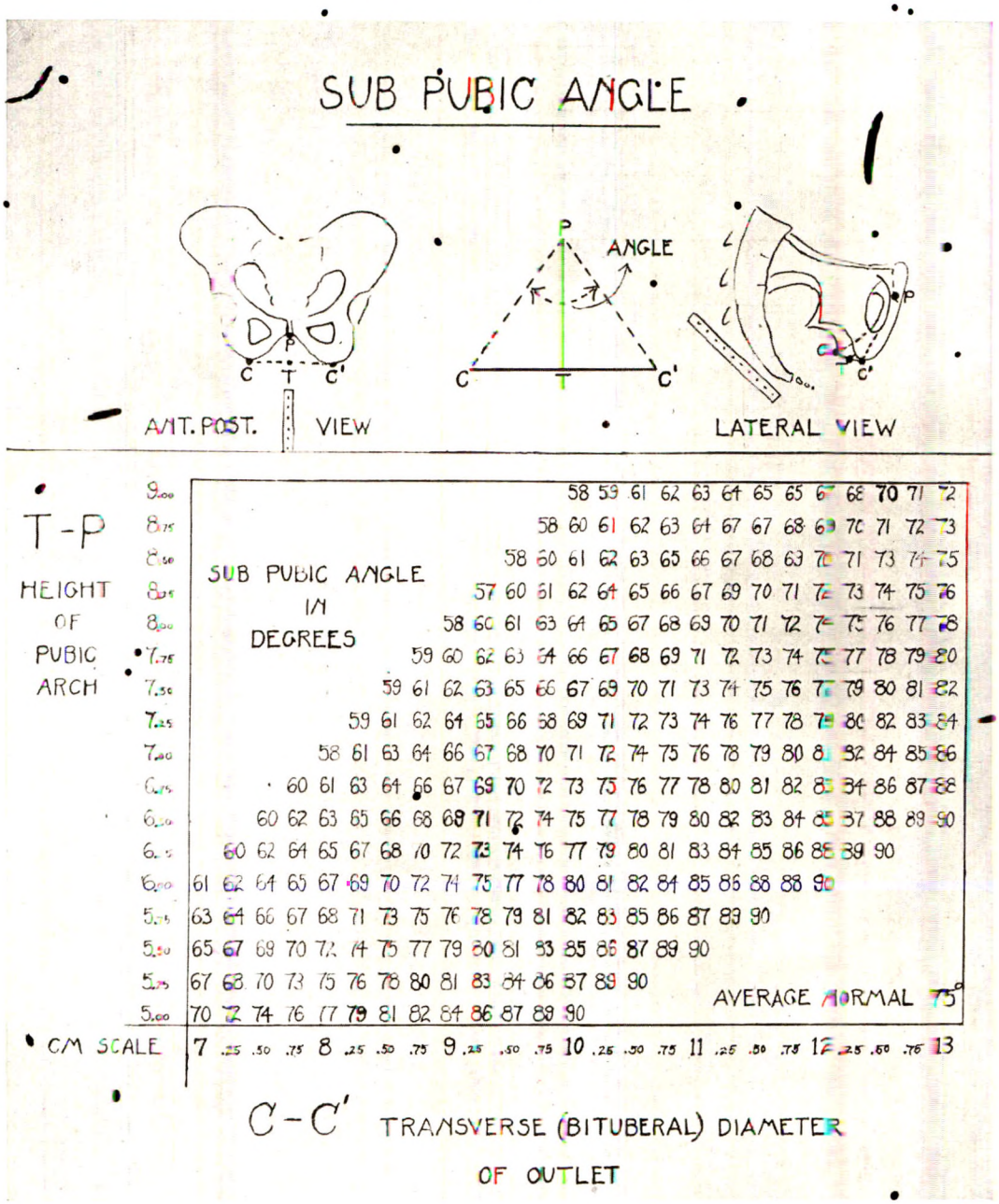


FIG. 7. A new method with a precalculated table for accurately measuring the subpubic angle. May be used with other techniques. The height of the subpubic arch is measured on the lateral view from the mid-tuberal point, *T*, to the lower inner symphysis, *P*. The base line of the subpubic arch, *C–C'*, is measured on the anteroposterior view. Where these two measurements meet in the table is the subpubic angle in degrees.

or another. I am impressed with its practicability.

I think it remains for every office or department of roentgenology to stick to one method that has proved practical in its experience and learn to use it well, being always mindful of the dangers that might be forthcoming from putting too much reliance on measurements alone. Exact measurements are quite all right, provided the data obtained are properly evaluated. A proper clinical approach is always necessary.

I would like to say a word about the figure given by Dr. Snow and quoted by the authors relative to the number of cases of serious arrest of labor. Eight to 10 per cent is given as the usual figure by several authors in cases of arrest due to cephalopelvic disproportion. This, I think, should be broken down a little in order that it be not misunderstood. For example, at the present time, the results at the Sloane Hospital show cases of serious arrest broken down into a cesarean section rate of 4.4 per cent and a 5.0 per cent rate of mid-forceps operation, making a total of 9.4 per cent. Of this 4.4 per cent of cesarean section, about 2 per cent is due to absolute disproportion which can be recognized without the use of measurements at all. One glance at a roentgenogram corroborating an already suspected disproportion clinically, will tell one that section is indicated. The other 2.4 per cent will be border line and require careful analysis based on the amount of disproportion, the type, and the pelvic morphology. An additional 5 per cent of cases include the use of mid-forceps. In these cases a disproportion exists which, with the help of forceps, will make the delivery from below relatively easy. It is in this group where a knowledge of the pelvic type, as well as the border line disproportion is important. The border line disproportion becomes increasingly important as the dangers inherent in a bad pelvic type are found to be present. Moreover, we must always keep in mind the varying extra-osseous factors which may compensate for minor disproportion. A border line situation is a more important factor in a forty year old primipara than in a young multiparous patient with no history of dystocia.

The point I wish to make is that in this total of 9.4 per cent of serious arrests, about 7 per cent will be a problem of careful correlation of morphology and size, and, it is in this group where the roentgen examination can be of real value *but* it is not going to be a matter of 2 mm. one way or the other as regards size of head and pelvis.

If we as roentgenological consultants are to keep the clinician interested in the diagnostic advantages of roentgenography in obstetrics, we will have to combat any tendency to regard its use as a rule of thumb procedure and instead attempt to emphasize its broader aspects, which combines a knowledge of the accurate pelvic diameters with the other known factors, both roentgenologic and clinical. If we do not do this, any procedure we popularize will easily lead us into error and place the roentgenologic aid in disrepute. I mention this only to emphasize what Dr. Colcher has already said.

The authors' method of getting the greatest transverse diameter and the bituberous on the same plane is very ingenious and they are to be congratulated on having worked this out. However, recent work has emphasized the advantages of pelvimetry in the erect position, both lateral and anteroposterior. This is particularly good for determining whether the head dips into the inlet. At least the lateral view will probably be desirable and it would be of advantage if the method under discussion could in some way be applied to this view. Otherwise, in my opinion, it should be added to the other roentgenograms obtained.

Dr. Colcher has emphasized the fact that the real level of the pelvic inlet is below what has always been considered the true conjugate. This we have recognized for some time and I am glad he has emphasized it.

Mention was made of the ease of pelvic typing when certain measurements are available. While this is quite true, we, in our department, have found that nothing quite supplants the ability which the stereoroentgenogram allows to study the pelvic image as a composite whole. It goes without saying, ever though I had a part in developing the stereoscopic method, that the added expense which the method entails makes it rather expensive for the average office, unless the volume of work is large enough to warrant its use, and unless the personnel becomes experienced in the stereoscopic technique of measurement.

Again let me commend the essayists for their contribution. The method is apparently practical and I hope to be able to give it a thorough trial.

DR. HARRY D. EVANS, Philadelphia. I have been partial to Snow and Lewis' method, for in my hands I have found it to be accurate and not too complicated. Recently, through the courtesy of Dr. Colcher, I have had the opportunity to use his ruler in 6 or 7 cases upon which I compared the results with those of measurements by Thoms' technique and also that of Snow and Lewis. I am happy to report that the greatest discrepancy noted between Dr. Colcher's method and the other techniques mentioned was never in excess of 3 mm. When we consider that errors up to and including 5 mm. are universally accepted in this kind of work, the results emphasize the accuracy claimed by Dr. Colcher.

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CLEIDOCRANIAL DYSOSTOSIS*

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CLEIDOCRANIAL dysostosis is a comparatively rare congenital defect of the bony skeleton, less than a hundred cases having been reported in the literature thus far. It is a syndrome in which the chief characteristic finding is a complete or partial absence of one or both clavicles. As so often happens, when one congenital defect is found in the body, another may also be found. In this condition one of the more constant defects accompanying that of the clavicles is a variation in the development of the skull bones.

Morand, in 1766, first described a case, which is not considered typical. In 1857, Stahmann reported a case of congenital absence of the left clavicle in a boy aged nine years. Barlow in 1883 reported a case of congenital absence of both clavicles and malformation of the cranium in a girl aged two years, probably the first typical case described. In 1897 Marie and Sainton reported 4 cases and applied the term "hereditary cleidocranial dysostosis" to the anomaly syndrome. Fitchet[2] in 1929 made a complete survey of all reported cases in the literature, summarizing each report. He stated that the chief features of this condition, as listed by Abrams from Marie and Sainton's article, are grouped under four headings as follows: aplasia, more or less marked, of the clavicles; exaggerated development of the transverse diameter of the cranium; delay in the ossification of the fontanelles; hereditary transmission.

There is much variation in the occurrence and degree of changes under the above headings, every case recorded not demonstrating all these findings. The anomalous development of one or both clavicles is the really characteristic defect. Therefore, Fitchet believed the term "hereditary cleidocranial dysostosis" should be discontinued and "congenital cleidal dysostosis" substituted instead, since the condition may be either hereditary or non-hereditary, with or without other anomalies. However, common usage will probably continue the designation of this condition by the older terminology.

The clavicular deformity may vary from defects at the acromial end to complete absence of one or both clavicles. The latter finding is very unusual, however. Most frequently, it is the middle portion of the clavicle that is involved, and the defect may vary from that of a narrowing to a thin bony or cartilaginous bar to the presence of only a ligamentous structure.

The skull defects may consist of brachycephalic changes, prominence of the frontal and parietal bosses or delay in closure of sutures and fontanelles. In 1907, Hultkrantz, making a survey of all cases reported up until that time, summarized by Fitchet, described the most characteristic skull finding to be the disturbed suture formation. These disturbances were explained to cause most of the other skull deformities. Kelley[3] described the skull as showing a globular, distended cranial vault with a bulbous forehead. He noted that the face is small in many of these cases, and that the suborbital region is depressed and sunken.

Regarding the familial and hereditary aspects, Marie and Sainton, according to Fitchet, stated that the condition had never

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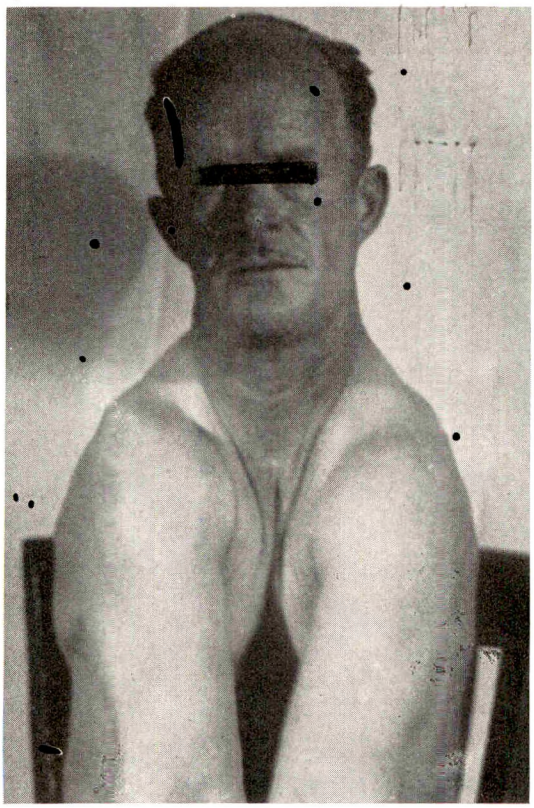


FIG. 1. Approximation of shoulders to midline anteriorly.

been known to run more than two generations, there being a tendency to revert to the normal type. However, later investigators have reported cases to extend through three and even four successive generations. There are also many cases reported that give no familial or hereditary history. On the basis of this fact, Fitchet divided the cases into two types: those with a familial and hereditary history and those without. Probably more of the cases come under the head of those giving no familial or hereditary history.

There have been other changes accompanying the clavicular deformity reported in the literature. Most of the patients are described as being of small stature. Anomalies of dentition are common, consisting of delay in the appearance of permanent teeth, incompleteness or irregularity of the second set of teeth, a proneness to caries, missing teeth or supernumerary teeth. A high palatal arch is also quite frequent. Defects in the bones of the hands and feet, including shortening of the toes, have also been reported.

The following case is being reported as meeting most of the requirements for classification as cleidocranial dysostosis, as set down by various authors in the literature.

REPORT OF CASE

T. H., male, aged forty-four, a veteran of World War I, was noted by routine physical examination and chest roentgenography at the Veterans Administration Facility at Biloxi, Mississippi, to have a deformity of the clavicles. Besides the anomalous findings, chronic bronchitis and a psychopathic personality with pathological emotionality were evident. There was no history or evidence of venereal disease.

The patient said that he knew from childhood of his clavicular deformity. It was discovered by Army physicians at the time of physical examination prior to his war service. As a consequence, he was placed on limited duty in the non-combat section and served from August 30,

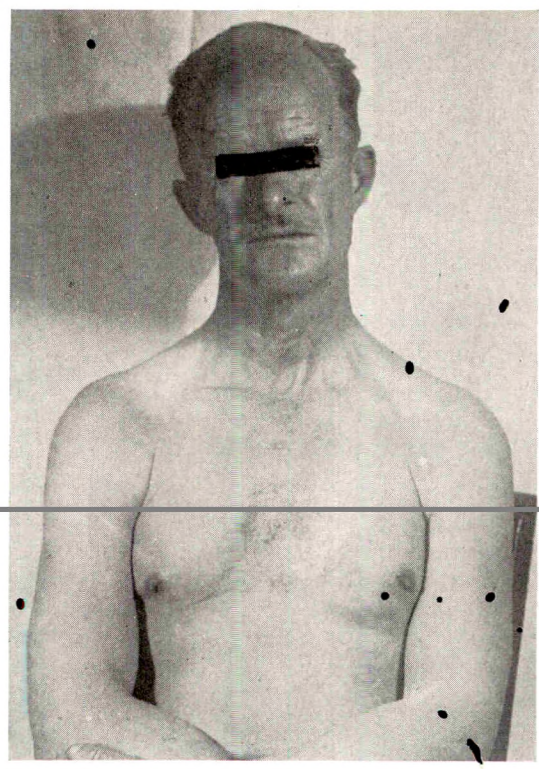


FIG. 2. Normal position of shoulders.

1918, to January 22, 1919, as a private in a developmental battalion.

Questioning as to a hereditary or familial history of this defect revealed that he had a brother, two years older than he, who had an absence of the left clavicle. This brother died in 1925. He recalled no similar defect in any other member of his family. The remaining family history and past personal history revealed nothing remarkable.

With respect to the clavicular deformity, it was noted on physical examination that he

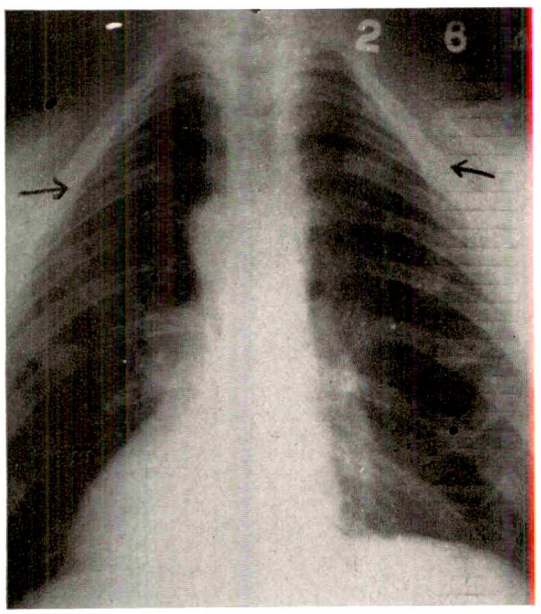


Fig. 3. Arrows point to rudimentary clavicles. Evidence of chronic bronchitis is also displayed.

carried his shoulders forward, like one who has suffered a bilateral clavicular fracture. Examinations of the clavicles revealed only a small, thin nubbin of bone, extending outward from the sternum on either side, each measuring about 1 cm. in width and 7 cm. in length. These pieces of bone were freely movable, and manipulation caused no discomfort. The shoulders were extremely mobile, much more than normal, and could be made to approach the midline anteriorly until they were only 5 to 6 cm. apart. The entire upper thorax was conical in shape. The muscles of the shoulder girdle appeared very well developed. He had a large head, globular in form, with prominence in the frontal and parietal regions. The face appeared small, in comparison with the remainder of the head.

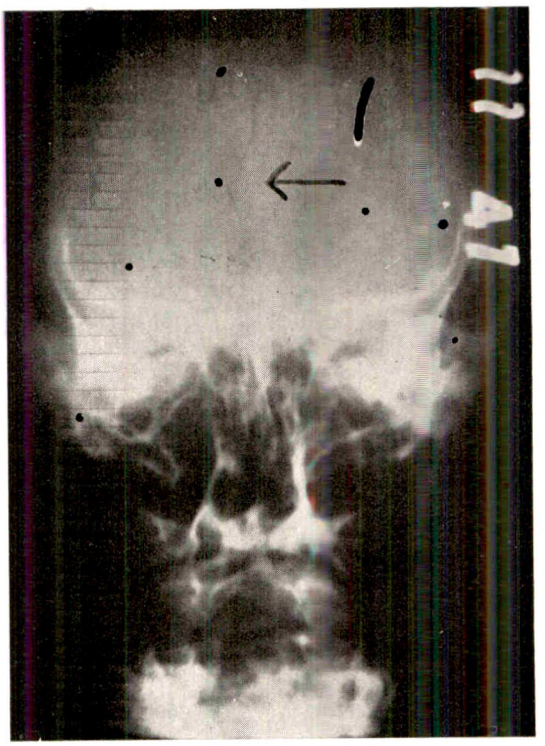


Fig. 4. Metopic suture line.

There was a depression in the malar region on both sides. All the teeth were missing. He gave



Fig. 5. Fingers, left hand, showing shortening of middle and distal phalanges and foreign body embedded in proximal phalanx, index finger.

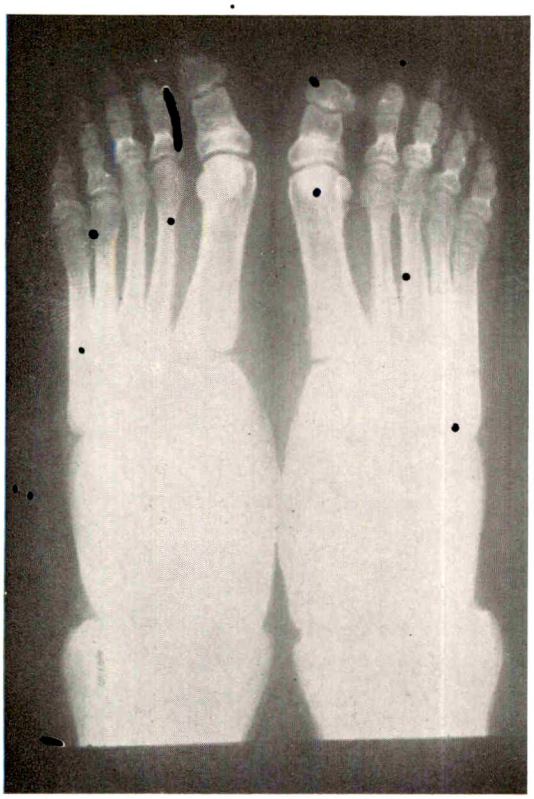


FIG. 6. Shows marked shortening of middle and distal phalanges of all toes.

a history of having had very poor teeth for years before they were extracted in 1930. He said that at the time of total extraction four supernumerary teeth were discovered in the upper jaw and were removed. He had a high, markedly large palate. It was further noted that the fingers of both hands appeared short and stubby and that the feet were small and the toes short. He said he wore size 4½ shoes. His height was 67 inches and his weight 141 pounds. His whole stature appeared somewhat slight.

Roentgen examination of the chest revealed a congenital deformity of the clavicle bilaterally. All that could be seen of the clavicles was a small bony nubbin extending outward from either side of the sternum in what would normally have been the position of the clavicles. These bony shadows measured approximately 7 cm. long and 1 cm. wide. The structure of the bone appeared normal. The remainder of the clavicles was entirely absent. The lung fields displayed evidence of a chronic bronchitis. Roentgenograms of the skull showed a somewhat globular shape of the cranial vault and a persistent frontal or metopic suture. The facial portion appeared small in relation to the remainder of the skull. The roentgenograms of the hands displayed a shortening of the middle and distal phalanges bilaterally and evidence of a foreign body within the proximal phalanx of the fingers of the left hand. Roentgen examination of the feet displayed a marked diminution in size of the distal and middle phalanges, especially the distal ones of the first toes, which appeared to be only about one-half normal size.

A roentgenogram taken of the right mandible following complaint of a small, tender swelling along the lower border of this bone revealed the presence of a supernumerary tooth in the anterior molar region. The tooth appeared to be embedded within a dentigerous cyst, and it was noted that the root of this tooth appeared to protrude slightly below the lower border of the mandible.

The patient also complained that it was difficult for him to sleep on his side. He stated that the upper shoulder would fall forward and impede his breathing. He was unable to lie on his back comfortably because his bronchial condition would cause him to cough. He therefore wore a special type clavicular brace which held his shoulders back and afforded him more comfort.



FIG. 7. Right mandible, showing supernumerary tooth embedded in dentigerous cyst with its root protruding below the lower margin of the bone.

The outstanding clavicular deformity, accompanied by a definite familial history and the presence of dental defects, skull defects and deformity of the hands and feet, seems definitely to warrant a diagnosis of so-called cleidocranial dysostosis.

The authors are indebted to Mr. S. C. Hemmings for the reproductions in this report.

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REGRESSION OF BONE METASTASES FROM BREAST CANCER AFTER OVARIAN STERILIZATION*

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AN EXTENSIVE literature has accumulated in recent years stressing the importance of ovarian sterilization in the treatment of metastatic lesions from cancer of the breast.[2,3,5,11,14,15,21,22] Local recurrences and deep, visceral metastases generally seem to show little or no response after radiation treatment directed to the ovaries, although occasionally very striking benefits in cases of these types are recorded in the literature.[4,7,8] However, the most satisfactory results seem to occur in patients with osseous metastases and several instances in which very definite and marked improvement of metastatic lesions had taken place have come to our attention. The purpose of this communication is to stress again the value of this form of therapy and to record a series of cases from the Pondville Hospital which have benefited from roentgen ovarian sterilization.

The rationale of the method is well established and the opinion of many authors is that, in properly selected cases, there is definite value in this mode of therapy.[6,12] The absence or withdrawal of the ovarian hormone is apparently the important factor in producing the favorable results.[10,13,18,20] In many instances, roentgen treatment directed to the pelvis with consequent sterilization has resulted in partial or complete recalcification of metastatic bone foci far removed from the area exposed to irradiation with no therapy directed to the local lesions. The question as to whether this form of treatment results in appreciable prolongation of life is still in doubt and cannot at present be definitely answered in the affirmative as the evidence is extremely difficult to evaluate and the conclusions of different authors are not in agreement.[9,16] It is our belief that there has been unquestionable prolongation of life in some instances, while in other cases there has apparently been no appreciable improvement in this respect.

There are, however, certain definite benefits which may be expected in properly selected cases. The chief among these can be summarized briefly as follows: (1) there is usually prompt and effective relief of pain; this may be of considerable duration and frequently lessens or temporarily obviates the need for morphia and other habit forming narcotics; in some instances, roentgen therapy abolishes pain when sedatives have failed to give relief; (2) the lesions in the bones regress partially and in some instances entirely reossify to disappear completely; (3) fractures with their resultant deformities and prolonged disabilities may be prevented; (4) there is great improvement in the patient's general condition with increased appetite and gain in weight; (5) there is marked prolongation of the patient's usefulness, many being able to continue their regular occupations for considerable periods with consequent benefit not only to the individual but to the family and the community; (6) there is striking betterment of the patient's mental status with helpfulness and optimism replacing the feeling of hopelessness and despair which so commonly occurs.

The technique of roentgen sterilization varies in its details in various clinics, but in general follows fairly well established rules. The object is to produce the desired result without injury to the skin and with as little disturbance of the patient through roentgen sickness as possible. In our clinic, the total dosage is usually 2,000 to 3,600 r, varying with the size of the patient. Two to four fields, each measuring about 15 by

* From the Pondville Hospital (Massachusetts Department of Public Health), Walpole, Mass. Presented at the Forty-third Annual Meeting, American Roentgen Ray Society, Chicago, Ill., Sept. 15–18, 1942.

15 cm., are used over the lower portions of the abdomen and lower back, the rays being directed into the pelvis. The filtration is 0.5 mm. of copper and 1.0 mm. of aluminum, giving a half-value layer of 1.0 mm. of copper. Two hundred kilovolts is the usual voltage and the target-skin distance is 50 cm. One field is exposed daily in rotation and the customary single dose is 200 r, although it may safely be increased to 250 or 300 r. With the larger dosages, a permanent sterilization usually results and in most instances is followed by hot flashes and other symptoms which may accompany the menopause. In earlier cases, total doses of only 1,200 to 1,800 r were administered with apparently satisfactory results. With smaller amounts of radiation the periods may return after an interval of six to eighteen months, which is undesirable and may necessitate another course of treatment. With our present therapy, there is little or no general reaction to the treatment and no permanent skin damage results. Occasionally there is nausea, malaise, loss of appetite or diarrhea, in which instance treatment may have to be suspended for a few days. Forcing fluids, giving large amounts of fruit juices and the administration of vitamin B complex usually control the untoward effects.

Some authors have favored routine ovarian sterilization of all patients with cancer of the breast in the premenopausal age periods.[17,19] It is our belief that in the light of present day knowledge, this is not advisable. Ahlbom,[1] in a very detailed study, showed that no definite benefits either as regards prolongation of life or the lessening of the incidence of metastatic lesions occur after ovarian sterilization. Moreover, patients suffering from cancer of the breast, particularly those who have been operated upon, should not have the added burden of a premature menopause unless it is reasonably certain that definite benefit is to be expected from the procedure. It is our experience that while ovarian sterilization has been followed by regression or total disappearance of osseous metastases, it has not prevented new metastatic lesions from appearing. In 3 of our cases, discussed in greater detail below, metastases in the bones have disappeared after roentgen irradiation of the pelvis only at a subsequent date to have new areas of destruction reappear in different portions of the skeletal system.

It is our usual procedure to observe all patients with cancer of the breast carefully at frequent intervals after operation or radiation therapy. Roentgen search for metastatic foci is carried out routinely two or three times annually. This usually comprises roentgenograms of the chest, skull, spine, pelvis and upper femurs. Roentgen examinations of the other bones are also made if there is pain, swelling or other clinical indication. At the first evidence of the development of bone metastases, roentgen sterilization should be performed. Roentgen therapy directed to the involved areas may be administered simultaneously in an attempt to speed the regression of the lesions, although in some instances the metastases regress or disappear without such local therapy. The general consensus is that about one-third or more of the cases treated may be expected to benefit from this form of treatment. We have not attempted to present statistics on our cases as, in many patients, the disease was so far advanced at the time treatment was instituted that death ensued too soon to permit of proper evaluation. In numerous instances, however, the benefits after sterilization were so striking that, though only an occasional case responded favorably, the treatment would nevertheless be very much worth while. There are no contraindications to ovarian sterilization and it may be used whenever there is any reasonable hope of benefit. Since pregnancy is contraindicated in patients with metastatic lesions, this is an additional benefit incidental to sterilization. Patients who are markedly cachectic should receive smaller daily doses and have the treatments distributed over a longer period than those in good physical condition.

CASE REPORTS

Case I. M. E. G., female, white, aged thirty-five. In April, 1937, she noted slight retraction of the left nipple and three months later felt a lump in the breast. She first sought medical attention in October, 1937, at which time the tumor had increased considerably in size. On October 25, 1937, left radical mastectomy was performed. Pathologic report was carcinoma simplex with metastases in four of seven nodes. The following month roentgen therapy was administered, 1,500 r to the left supraclavicular area, the left axilla anteriorly, the posterior aspect of the left axilla, and the left chest anteriorly.

In November, 1938, there was no evidence of local recurrence. A mass was palpable in the right breast. The patient complained of backache, and roentgen studies revealed extensive metastatic disease in the ribs, spine, pelvic bones and upper femurs. Roentgen therapy was administered, 1,200 r to the pelvis anteriorly, 900 r to the right side of the pelvis posteriorly and a similar amount to the left side posteriorly. In January, 1939, she stated that she had had prompt relief of pain and was entirely symptom free; her last menstrual period had been on December 12, 1938. In August, 1939, she reported feeling entirely well and that there had been a gain of 18 pounds in weight since the last roentgen treatments.

Roentgen studies carried out February, 1940, showed filling in of the disseminated osteolytic metastases. The patient looked well and reported she had no complaints. In August, 1941, she stated that she had been symptom free until a very short while previously, although recently she had noticed increasing apathy, weakness and weight loss. On physical examination, there was evidence of marked cachexia and the picture was consistent with advanced generalized carcinomatosis. There was no local recurrence. The patient died on August 19, 1941, in uremia. Permission for postmortem was not obtained.

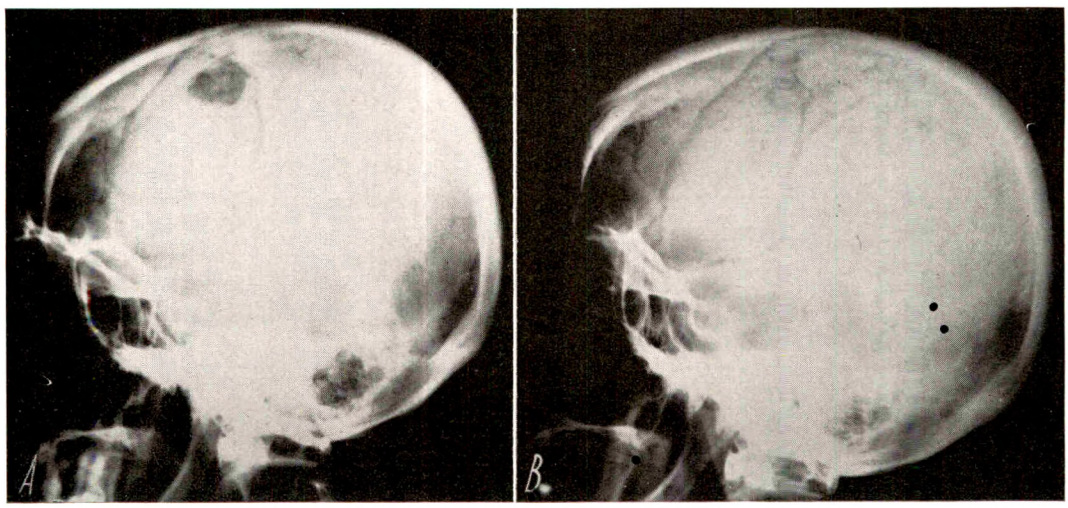


Fig. 1. Case II. *A*, roentgenogram of the skull, July 1, 1933, showing large, rounded area of radiance in the parietal region. Metastatic carcinoma. *B*, June 23, 1937, almost four years later. The metastatic focus in the parietal region seen in *A* is now evidenced by an area of slightly mottled density, having disappeared almost completely.

Case II. M. B., female, white, aged thirty-seven, married. In February, 1932, the patient felt tenderness in the right breast and a lump about the size of a walnut. In May, 1933, she showed advanced malignant disease involving the right breast with large masses of metastatic lymph nodes in the right axilla. She was treated with radium at that time, receiving 6,720 milligram-hours, with sixteen needles of 2 to 3 mg. each, distributed throughout the breast and an additional dose of 3,360 mg-hr. inserted directly into the main mass in the breast. Roentgen studies on July 1, 1933, revealed metastatic cancer involving the skull with an area of destruction measuring about 2 cm. in diameter in the parietal region on the left side (Fig. 1*A*).

In October, 1933, sterilization by the roent-

gen method was performed, the dosage being 600 r to an anterior and 600 r to a posterior pelvic field, each 10 by 15 cm., with 200 kv. and 0.5 mm. copper filtration; 1,200 r was also delivered to the right chest anteriorly. The patient was observed at intervals thereafter and the primary disease apparently remained under control. The lesion in the skull, described above, filled in completely and roentgen studies on March 12, 1936, and again on June 23, 1937, showed no evidence of recurrence in the skull (Fig. 1*B*). In August, 1936, she began to have left frontal headaches and failing vision in the left eye. Roentgen therapy to the head, left frontal area, was administered in July, 1937, 1,200 r being given with 200 kv. and 0.5 mm. copper filtration. The patient died on February 10, 1939, approximately seven years after her first observation of the mass in the breast, of disseminated metastatic cancer.

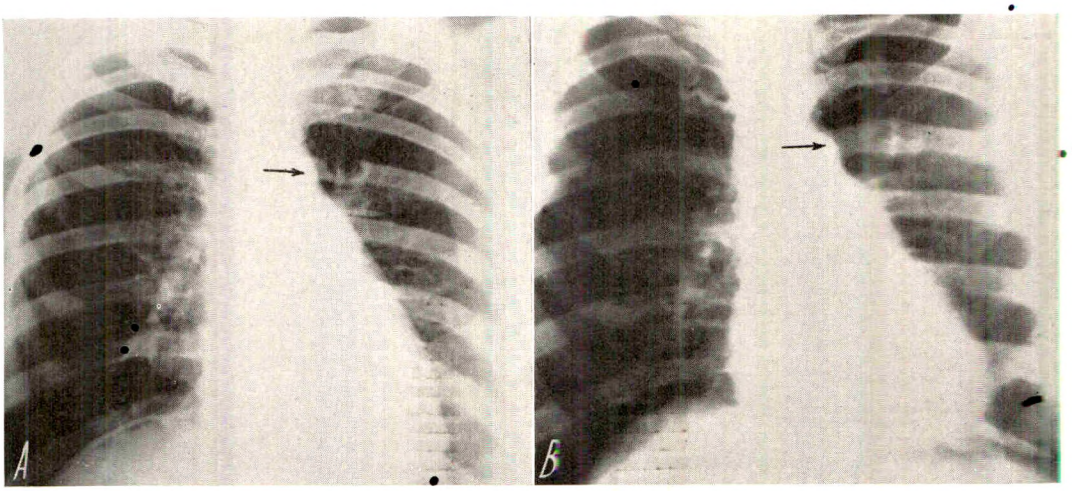


FIG. 2. Case III. *A*, July 13, 1940. There is a metastatic lesion in the posterior aspect of the sixth left rib with a large rounded area of destruction in the paravertebral portion of the rib. *B*, same patient nearly two years later on June 29, 1942. The lesion in the sixth left rib has reossified almost completely.

CASE III. L. A. R., aged thirty-nine, married. The patient noticed a lump in the right breast in 1935 and visited her physician but was not referred for surgery till June, 1936, at which time a radical mastectomy was performed. The pathologic report was adenocarcinoma with axillary metastases. One year later, there was no evidence of local recurrence. A node was palpable in the left axilla. The patient complained of pain between the scapulae. Roentgen studies of the chest, skull, spine and pelvis revealed no evidence of metastases. In March, 1938, she still complained of back pain; there was no tenderness or weight loss. Roentgen studies again were negative.

The patient was seen periodically about three times annually and in July, 1940, metastatic disease was demonstrated in the ribs, left ilium, skull and lungs (Fig. 2*A*). In October, 1940, roentgen sterilization was performed, the dosage consisting of 400 r each to right and left anterior pelvic fields and 600 r each to the right and left posterior pelvis. The factors used were 200 kv., 0.5 mm. copper filtration and 10 by 15 cm. fields. Three months later, the patient was entirely symptom free except for frequent hot flashes. The metastatic area in the pelvis had filled in; the lesion in the skull was definitely smaller, and the destructive process in the ribs was less marked than previously. In February, 1941, the patient received 1,600 r over the right chest. When seen a few months later, she was symptom free. In April, 1942, the metastatic areas in the skull, ribs and pelvis had disappeared almost completely and the patient looked and felt well (Fig. 2*B*).

CASE IV. R. L. V., female, white, aged forty-one, married. In 1928, she first noted the appearance of a small lump in the left breast and the following year a left radical mastectomy was performed. No report of the microscopic findings could be obtained. There was wound sepsis and abscess formation after this operation. In 1931, she was operated on for recurrent tumor, the pathologic report of which was as follows: "a mass of glands measuring [illegible] by 2 cm. showing

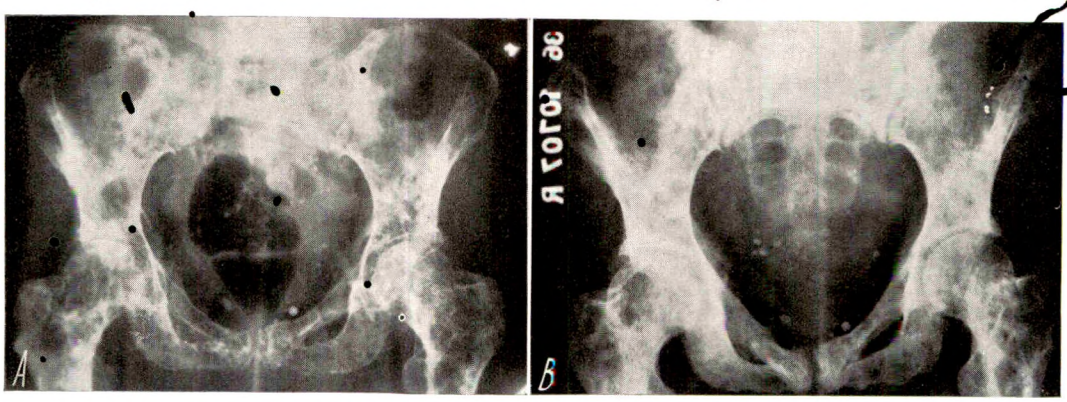


Fig. 3. Case v. *A*, April 15, 1936. There are advanced osteolytic changes involving the lumbosacral region, the bones of the pelvis and the upper femurs. *B*, September 9, 1936. There has been extensive proliferation of new bone with marked regression of the metastatic lesions.

metastatic carcinoma," suggesting that the previous operation had been incomplete. Three years later, a mass about 3 cm. in diameter recurred in the mid-portion of the operative scar and this was removed with the high frequency knife. The wound was not closed and did not heal. Seven months later, a large ulcerative tumor about the size of an egg was removed in the region of the previous excision. The pathologic report was "rapidly growing adenocarcinoma."

The patient entered Pondville Hospital in September, 1935, with recurrent carcinoma involving the left chest wall in the region of the operative wound and left axilla. A biopsy was performed, the report being returned as carcinoma simplex. Roentgen studies revealed early metastatic disease with extensive destruction of the body of the left scapula, the upper third of the shaft of the left humerus, and the ribs. The patient was given roentgen therapy, 1,200 r being administered to the front and back of the left chest. Sterilization was performed in October, 1935, 600 r being delivered to the anterior pelvis and a similar amount to the pelvis posteriorly. The kilovoltage was 200, with filtration of 0.5 mm. of copper and 1 mm. of aluminum over a field 15 by 15 cm. In November, 1935, a total dose of 900 r was delivered to the left chest anteriorly. The patient was seen a month later and felt well. There were no palpable masses. The skin in the left axilla showed extensive erosion.

In July, 1936, she returned for observation. A firm mass was palpable in the right breast and there were numerous enlarged lymph nodes in the right axilla. The metastatic lesions in the bones, above described, showed practically complete regression with extensive new bone formation in the humerus, scapula, and ribs on the left, these areas now appearing practically normal on the roentgenogram. The patient showed evidence of much weight loss. Death ensued on September 27, 1936. Permission for autopsy was not obtained.

Case v. M. L. B., female, aged forty-three, married, no pregnancies. Her menstrual history had been essentially normal, although the periods had been slightly irregular and the flow had decreased slightly during the past year. In October, 1933, she first discovered a lump in the right breast and three days later the breast was removed. Six roentgen treatments were given postoperatively and a few months later three more roentgen treatments were administered. Two years later, she began suffering from pain in the back which became progressively more severe. In April, 1936, she entered Pondville Hospital. There was no local recurrence on the right, but a neoplasm was present in the left breast. Roentgen studies revealed extensive skeletal metastases with multiple destructive lesions in the skull, spine, ribs, pelvis and femurs (Fig. 3*A*). Sterilization was performed. The dose given was 900 r to an anterior and posterior pelvic field with 200 kv. and 0.5 mm. copper filtration. In June, 1936, she was quite comfortable and was able to sit up. Roentgen studies on September 9, 1936, revealed marked improvement of the bone lesions with extensive filling in and much new bone formation (Fig. 3*B*). The changes were particularly striking in the skull, the areas of destruction being very

markedly decreased both in number and extent. Through 1937 and early 1938 she felt entirely well and was able to be up and about. In July, 1938, the patient stated she was losing weight rapidly but continued very active. In September, 1938, the patient committed suicide.

CASE VI. R. P., female, aged forty-four, married, two children living and well. In 1939, she had a right radical mastectomy for cancer of the breast. When seen in July, 1940, she showed two recurrent nodules in the scar and stated that these lumps had been present for several months and had been gradually increasing in size. Roentgen therapy was administered with 200 kv. and 0.5 mm. of copper filtration; 3,000 r was delivered over each nodule. Roentgen studies of the chest, skull, pelvis and spine revealed no evidence of metastases. Three months later she returned complaining of backache. Roentgen examination showed metastatic involvement in the neck of the left femur and the inferior portion of the right ilium slightly above the acetabulum. Roentgen sterilization was performed, 1,200 r being administered to each of four pelvic fields, two anterior and two posterior. Also, 1,600 r was delivered to the lumbar area, the left chest posteriorly and the anterior surface of the right shoulder.

When seen in April, 1941, she was entirely free of pain and felt well. In November, 1941, she had a productive cough. Roentgen studies showed the metastatic lesions in the bones filled in. She died in January, 1942. No autopsy was obtained.

CASE VII. R. M., female, aged forty-seven, single. Menstrual history uneventful. In June, 1931, she noted a mass about as large as a cherry in the upper portion of the left breast, but did not consult a physician till February, 1933. In June, 1933, she was admitted to the Pondville Hospital with massive involvement of the left breast by scirrhous carcinoma. There was ulceration of the medial aspect of the breast with multiple skin nodules extending in every direction from the area of ulceration. No masses were present in the axilla and there was no edema of the arm. There was fullness in the left supraclavicular fossa. The right breast and axilla were normal. Roentgen studies revealed extensive osteolytic metastases in the skull, the pelvis, and the upper ends of the femurs (Fig. 4*A*), the vertebrae and the shoulder girdles. There were extensive, bilateral pulmonary metastases. Roentgen treatment with 200 kv. and 0.5 mm. copper filtration was administered; 1,200 r to the left breast, 800 r to the pelvis posteriorly and 600 r to the anterior pelvis. During the next two months she remained in bed and appeared to improve slowly with gain in weight and strength. She suffered mild attacks of dizziness with nausea and vomiting, considered to be due to the cranial metastases. In February, 1934, roentgen examination revealed marked regression of the

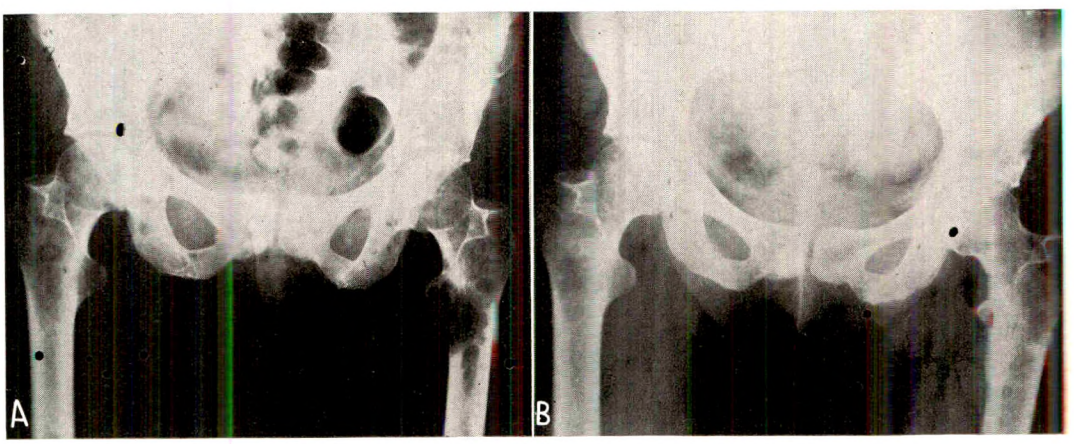


FIG. 4. CASE VII. *A*, June 8, 1933. Advanced metastatic carcinoma with multiple areas of destruction in the bones of the pelvis and upper femurs. *B*, December 6, 1934. There has been extensive recalcification of the osteolytic lesions, many of which have disappeared completely. The right ischium and ilium show a return to practically normal contour and bone structure

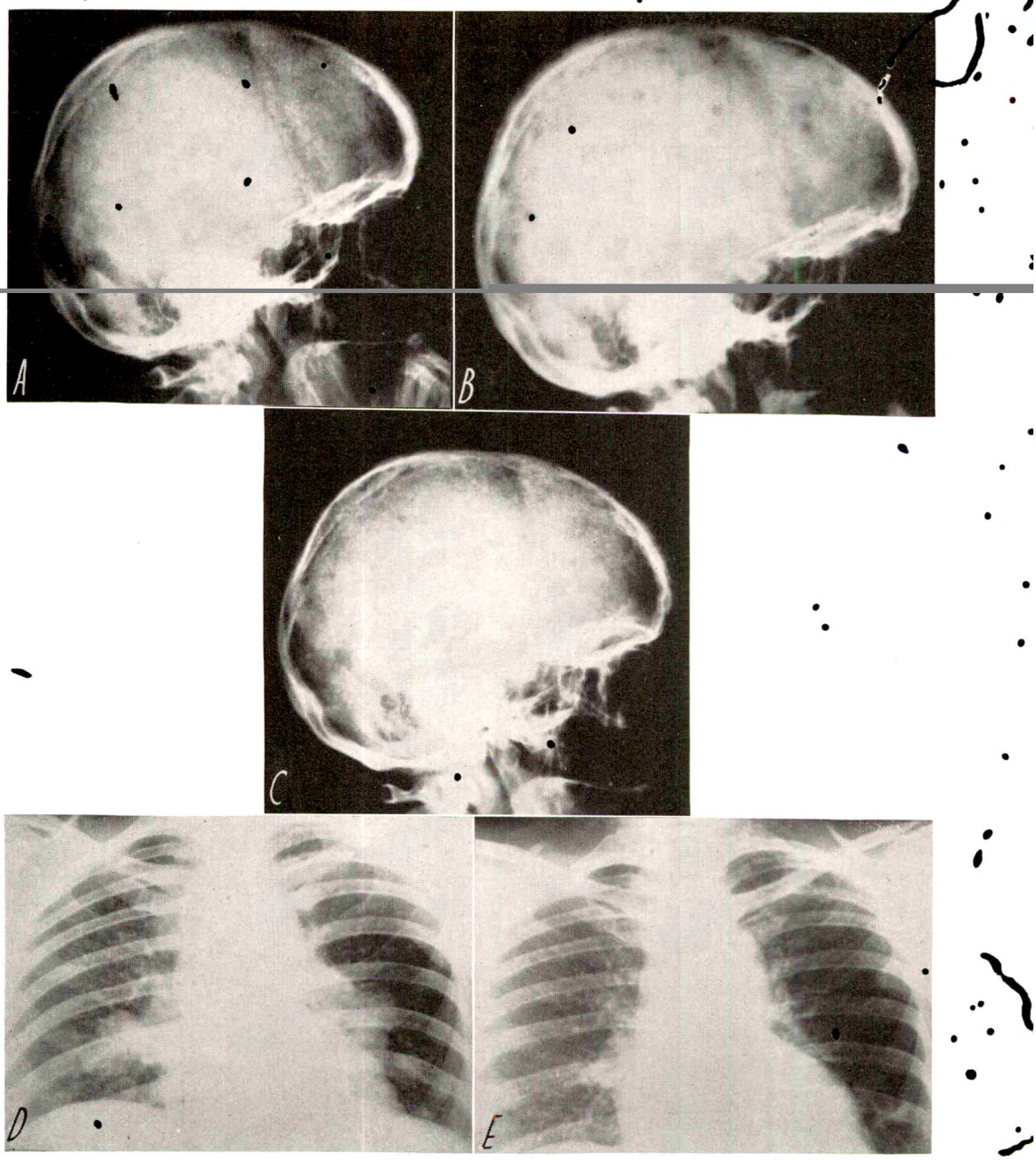


FIG. 5. Case VIII. *A*, February 16, 1933. Several very small foci of metastatic carcinoma are demonstrable in the frontal and parietal regions. *B*, July 20, 1934. There are multiple metastatic lesions scattered throughout the skull. *C*, December 28, 1934. The areas of increased radiance in the skull have decreased markedly in number and size. *D*, February 19, 1933. Advanced metastatic carcinoma involving the right lung, the left base, the mediastinum and the hilar glands. *E*, December 28, 1934. There is now no definite evidence of pulmonary metastases.

bone lesions with extensive regeneration of new bone. Follow-up studies six months later showed further improvement of the bone lesions. In December, 1934, the bone metastases were still well filled in (Fig 4*B*). Her general physical condition had improved further and she had gained 8 pounds.

In March, 1935, roentgen studies showed re-

currence of the bone metastases. Pain had recurred, especially in the chest and thighs, and she was confined to a wheel chair. Radiation therapy was administered to the left breast, the right shoulder posteriorly and the lumbosacral region. The pain increased and in August, 1935, she suffered a spontaneous fracture of the left femur. The patient died on September 21, 1935. Postmortem studies revealed extensive metastatic disease in the left axilla, chest, liver, adrenals, spleen, ovary and the skeleton.

Case VIII. H. S. A., female, aged forty-six, married. Her menstrual history was uneventful and she was still menstruating when first seen at the Pondville Hospital. In March, 1932, she had a left radical mastectomy because of a slowly growing mass which she stated had first been noticed four years previously. In January, 1933, there was a rapidly growing nodule overlying the upper sternum, and the following month she entered the Pondville Hospital. Roentgen studies revealed bilateral pulmonary metastases (Fig. 5*D*); there were several small areas of increased radiance in the skull (Fig. 5*A*). Roentgen treatment was given, 1,600 r being delivered through a field 15 by 15 cm. over the lesion on the anterior chest wall. The mass disappeared completely within the succeeding few months, but the pulmonary metastases increased and in June, 1933, 12,000 r was delivered over a field 20 by 20 cm. on the lower thorax posteriorly.

In December, 1933, the patient complained of severe pain in the lower back and had a slight cough. Roentgenograms revealed extensive destruction due to metastatic cancer in the bones of the pelvis and the upper femurs. She received 800 r over the lower back with prompt relief. About six months later, there was a recurrence of severe pain in the back and legs. Two small, movable nodules were present in the left axilla. Roentgen studies demonstrated multiple punched-out, osteolytic metastatic lesions in the skull (Fig. 5*B*), and a slight increase in the destructive processes previously noted in the pelvis and femurs. In July, 1934, roentgen therapy was administered as follows: 800 r to the pelvis and lumbar spine and 1,200 r to the left pectoral region. There was considerable relief of pain following the treatments. When seen three months later, she stated that she had had no menstrual periods and was having menopausal symptoms. As there had been a recurrence of pain in the back, a single dose of 400 r was delivered over the pelvis posteriorly.

In December, 1934, she was entirely free of pain. Roentgen studies revealed marked regression of the bone lesions, the skull particularly showing great improvement (Fig. 5*C*). The lungs also appeared free of metastatic disease (Fig. 5*E*). She was observed at frequent intervals and her condition remained satisfactory with some weakness and discomfort in the lumbar region, but no pain. In 1936, a report was received from another hospital that she was suffering from cancer of the rectum, for which a colostomy was performed. Death occurred on June 11, 1938, and the autopsy report was "cancer of the rectum, cancer of the peritoneum, intestinal obstruction, generalized peritonitis, bilateral cysto-ureteral pyelonephritis."

SUMMARY AND CONCLUSIONS

Osseous metastases from cancer of the breast may regress or disappear completely after ovarian sterilization.

The absence or withdrawal of the ovarian hormone is the important factor in producing the results.

In addition to the beneficial effects observed in the bone lesions, there may also occur marked relief of pain, general improvement in the physical and mental status, and the prolongation of the patient's period of usefulness.

The regression of the osseous metastases prevents pathologic fractures with their resultant deformity and disability.

The question as to whether this mode of therapy results in appreciable prolongation of life cannot be definitely answered in the affirmative at present.

Routine roentgen sterilization of all patients with cancer of the breast is not advocated, the treatment being recommenced when osseous metastases have been demonstrated by roentgen examinations.

About one-third or more of the patients treated may be expected to show improvement after ovarian sterilization.

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DISCUSSION

Dr. C. M. Richards, San Jose, Calif. There is just one other horn of the dilemma that was not brought out in Dr. Ritvo's paper, although he may have mentioned it in his complete paper, and that is that once we have produced a menopause in these patients then we are faced with the dilemma of mitigating the menopausal symptoms. It is our experience that we have to be very watchful where the patient is also under the care of the family physician to see that he does not start giving the patient estrogenic substances to relieve her menopausal symptoms and thus put her back to the place where she is getting her estrogenic substances again, thereby defeating the object that we have obtained in producing the menopause. I think we must be very watchful of that or else our results will be vitiated.

Dr. Ira I. Kaplan, New York. I am very much interested in this paper because we meet with this question all the time in our work in New York. In the hospital in which I am principally interested, we naturally get the last stage cancer cases and frequently are requested to take care of patients who have extensive skeletal metastatic lesions. We treat them with sterilization whenever we can, but we are not quite sure whether we should or should not stop at the menopausal age because frequently, in spite of the fact that the patient has been through the menopause, if we still irradiate the pelvis over the ovarian areas in a number of instances we do still get recession of skeleton metastasis.

One of the discussants reported a very interesting case in which there was a resolution of the skeletal metastasis but no resolution of the liver metastasis. We find that in numerous cases. While the skeletal metastases respond to sterilization, the visceral metastases do not seem to so respond.

Now there is another important question

and that is the question of cancer of the breast, metastasis of the skeleton, and the presence of pregnancy. What shall we do in that type of case? Shall we interrupt the pregnancy? Shall we sterilize the patient thereby? Shall we interrupt the pregnancy with manual methods and then sterilize the patient or what should we do? That is one of the most important questions that we often encounter in our service.

Dr. Richards mentioned a very important point, which I think should be emphasized constantly, and that is the attempt of the physician to ameliorate menopausal symptoms by estrogenic injections. It just upsets everything you try to do with sterilization for metastatic cancer. It is a rule with us that when we return patients to the physicians or send them back to other clinics we distinctly tell them that estrogenic hormone treatment should not be instituted.

ROENTGEN IRRADIATION IN POLYCYTHEMIA VERA BY MULTIPLE SMALL DOSES TO LARGE AREAS OF THE BODY*

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ANY METHOD of treatment, particularly one which may be dangerous to the patient, should be checked and evaluated at intervals. It is the purpose of this paper to study a method of roentgen therapy for polycythemia by multiple small doses to large areas of the body, which has been used in the Tumor Clinic of the Massachusetts General Hospital over a ten year period, from 1932 through 1941. This study, confined as closely as possible to the point of view of the radiologist, has resulted in several interesting observations as well as in some modification of the technique of treatment.

In a brief review of the literature the development of roentgen therapy in polycythemia vera may be traced from 1907 when Stengel[5] was the first to advocate irradiation of the bone marrow for this disease. In 1916 Lüdin[5] first successfully irradiated the skeletal long bones of an erythemic patient, and for many years this proved to be the best method of radiation therapy. In 1927 Teschendorf[10] advised irradiation of the entire body for generalized lesions, and in 1932 Sgalitzer,[8] using this method, reported good results in polycythemia. Hunter[3] and Sanderson[7] in 1936 reported 3 cases which are included in the present series. Pierson and Smith[6] believe that this is a satisfactory method of treatment.

Irradiation with multiple small doses given to large areas of the body is the treatment of choice after the diagnosis of polycythemia vera is established. Before treatment, however, there are several points which the radiologist must consider. In the first place, the diagnosis must be definite. It is dependent upon a persistent polycythemia and, in the majority of cases, splenomegaly. Polycythemia due to chronic heart and lung lesions, as well as physiological response to high altitudes, must be eliminated as these should never be treated by irradiation. The percentage of oxygen saturation of the arterial blood is normal in polycythemia vera, whereas it is low in congenital heart disease and in pulmonary lesions. Bone marrow biopsy is of diagnostic value, particularly in the ruling out of leukemic polycythemia, but is seldom necessary.

Table 1 gives an analysis of 20 cases of polycythemia vera treated at the Massachusetts General Hospital during a ten year period. The dosage has been tabulated in round figures and represents the sum of the amounts given to the two fields.

METHOD OF TREATMENT

In the 20 cases under discussion, daily doses of 20 to 50 roentgens were used. Small doses were chosen because, being unlikely to cause roentgen sickness, they might be more valuable than large doses by prolonging the series of treatment. Total doses varied from 200 to 1,200 r per series, the majority of patients receiving 500 to 600 r per series. Because satisfactory remissions were secured from this dosage, it has been continued. The field covers the body from knees to neck. This is accomplished by using a 20 by 20 cm. cone on the machine and a target-skin distance of 215 cm. The field is centered at the level of the crest of the ilium, and alternate anterior and posterior fields are used. The other factors are 200 kv., 0.5 mm. Cu plus 1 mm. Al filter,

* From the Department of Radiology, Massachusetts General Hospital, Boston. Presented at the Forty-third Annual Meeting, American Roentgen Ray Society, Chicago, Ill., Sept. 15–18, 1942.

giving a half-value layer of 0.92 mm. Cu. Calculation of the output of the machine is determined with the Victoreen r meter, measured in air, at the actual target-skin distance and by the inverse square law. The present daily dose is 20 to 30 r, for a total of approximately 500 r in one series; that is, 250 r anteriorly and 250 r posteriorly.

Weekly white blood counts are done until a normal range is reached; then they are checked daily. A fall in the white blood cells below 4,000 or 5,000 warrants an interruption of treatment. During the period of treatment the red blood count and hemoglobin do not, as a rule, change greatly, but if at the end of two or three months the red blood count has not fallen satisfactorily the course of treatment may be repeated cautiously.

RESULTS OF TREATMENT

Of the group studied, one patient died early in the treatment (Case 19); one death occurred six weeks after the completion of treatment (Case 6). One patient showed a temporary fall in the red blood count but seven months later a rapid rise occurred (Case 13). One patient has not been followed (Case 4). The remaining 16 patients did very well, showing remissions varying from six months to five years. Two of this group are dead of other causes. It is important to note that those with the longest remissions had a white blood count of 6,000 or less at the completion of treatment.

The patient usually showed no irradiation reaction and began to improve symptomatically while under treatment. Many continued their work during treatment. The white blood count remained low (1,400 to 6,000) for approximately a month after treatment and then gradually returned to normal. The red blood count began to decrease toward the end of the series of treatments and continued for several months at levels of 3.5 to 6.0 million. When the red count rises above 6.5 to 7.0 million the series of treatments should be repeated. The spleen may or may not regress in size, but usually does. The remission of symptoms is variable in length of time, but in our experience is longer on an average than with other methods of treatment.

COMMENT

The dangers of roentgen treatment by multiple small doses to large areas of the body are, as in other methods of irradiation, leukopenia and the possibility of late irradiation anemia. While leukopenia is present the patient may be unable to combat an intercurrent infection, as may have occurred in Case 6. Anemia may develop in untreated cases also and its appearance in treated cases is not necessarily due to treatment. Bleeding duodenal ulcers, for instance, are common in this disease and may cause severe anemia. In the group of cases studied no anemia developed which could be attributed to radiation treatment.

The primary advantage of this method, as compared with other forms of radiation therapy (that is, larger daily doses to the ribs or systematic irradiation of the long bones[4]) is that it gives longer periods of remission with less danger of irradiation damage to tissue. It is no more dangerous; it does not cause roentgen sickness as a rule; and it does not interfere with the patient's daily work. Irradiation of the spleen has no lasting effect either on the symptoms or on the red blood count (Case 1). Irradiation of the pylorus and Brunner's glands has not proved successful.[9] Early reports indicate that treatment with radioactive materials may be of value,[1] but until they have been tested more extensively and until their cost is lower and the supply more plentiful, roentgen treatment with multiple small doses will continue to be of more practical value. Phlebotomy gives good temporary relief, but must be repeated at relatively frequent intervals. Treatment with phenylhydrazine, its derivatives, or arsenic—toxic drugs whose use must be carefully controlled—is of value only while the drugs are taken, and they usually cause the patient marked discomfort.

TABLE I

ANALYSIS OF 20 CASES OF POLYCYTHEMIA VERA TREATED WITH MULTIPLE SMALL DOSES OF ROENTGEN RADIATION AT THE MASSACHUSETTS GENERAL HOSPITAL (1932–1941)

	Case	Sex	Age	Symptoms	Blood Count				Spleen	Date	Treatment	Oxygen Saturation per cent	Remarks	Length of Remission	Final Status
						Hg	Erythrocytes (million)	Leukocytes							
U-65050	1	M	52	Weakness; headache; bleeding ulcer	B	65% T	5.3	17,300	Enlarged	8/19/32	600 r spleen—each 3 series		No decrease in size of spleen		
					A		6.7	16,500	Enlarged	9/26/32					
					B	100%	10.6	17,400		6/27/33	500 r—30 r daily				
					A		10.3	7,300		7/20/33					
					B					9/25/33	1,200 r—50 r daily				
					A	75%	4.9	5,700		10/28/33				1½ yr.	
					B		5.7			7/22/35	180 r—30 r daily			2½ yr.	
									Enlarged	2/14/38	600 r spleen				
									Enlarged	8/14/38	600 r spleen				
					B	135% S	7.3	16,600		11/14/38	900 r—30 r daily			2½ yr.	
					A		5.6	4,300		3/—/39					
									Enlarged	9/16/40	600 r spleen				
					B		7.9			6/23/41	500 r—20 r daily				
					A	14.0 gm.	4.7	9,200		12/31/41				6 mo.	Working; in good health
10879	2	F	49	Headache; migrating phlebitis legs and abdomen	B		7.0	12,950	Enlarged	11/25/32	300 r—30 r daily		Red blood count still elevated		
					B		8.1			1/24/33	600 r—20 r daily				
					A	60% T	4.4	4,200	Negative	4/—/33				4 yr.	
					B	100% S	6.2	7,100		4/ 5/37	600 r—25 r daily		Recurrence of phlebitis; improved		
					A		4.5			7/—/37				3½ yr.	
						19.5 gm.	5.8	7,500		7/15/40			Became obese against advice		Sudden death after large meal; coronary; no autopsy
										11/—/40					
129760	3	F	38	Hypertension 300/100; kidney damage	B	100% S	7.3	17,000	Slight enlargement	8/30/32	600 r—20 r daily		Treatment after venesection of 1,300 cc.		
					A		3.7	1,400		10/17/32				5 yr.	
					B	164.0 gm.	6.0	11,300	Slight enlargement	1938	9200 r rt. breast and axilla—7 mo. period		Inoperable cancer (breast)		Died; cancer; cardiac failure
H-3	4	M	54	Paralysis rt. leg and arm; headache; aphonic	B	123% S	7.6	13,000		9/ 6/32	600 r—20 r daily		Venesections—800 cc.; no follow-up. In another hospital Sept., 1933; reason unknown		Unknown
					A		7.4	8,600		10/ 4/32					
30403	5	M	62	Vertigo 8 yr.; cerebral accident 6 mo. before; gangrene of toe on entry	B	103% S	6.6	11,000		4/21/36	400 r—30 r daily		Toe amputated		
					A		5.4	3,100	Negative	6/—/36				3 yr.	
					B		6.0			4/ 7/39	600 r—30 r daily				
					A	14.3 gm.	4.9	6,900		11/ 3/41			Improved and able to work	2¾ yr.	General condition has remained unchanged
							5.5								

(Key—B—Before; D—During; A—After.

Case	Sex	Age	Symptoms		Blood Count: Hg	Blood Count: Erythrocytes (million)	Blood Count: Leukocytes	Spleen	Date	Treatment	Oxygen Saturation per cent	Remarks	Length of Remission	Final Status
6	M	65	Swelling, discoloration feet and legs	B	130% S	9.9	17,000	Slight enlargement	12/ 8/36	300 r—30 r daily	91.8	Clinical diagnosis, deep thrombophlebitis and cellulitis		
5589				A		7.5								
				B					2/ 9/37	700 r—50 r daily		After treatment, lumbar puncture; discharged; 4 days later re-entry because of headache. Spiked temp. to 102° F.; frank hemoptysis. Chest x-ray: cavities; consolidation rt. upper lobe		Died 6 wk. after re-entry; no autopsy
				A		7.2	3,300 1,300 4,000		2/25/37 March '37					
7	F	50	Headache; plethora; blood pressure 178/128	B	122% S	8.1	9,500	Negative	5/ 9/37	500 r—30 r daily	95.5	Felt much improved	4½ yr.	Continues to feel well
40086				A		5.7	6,800							
					16.1 gm.	4.7	10,200		9/ 8/41			Feeling well		
8	M	62	Difficulty walking: unsteady gait						1934 1938	Roentgen treatment to chest for neurofibroma		Pathologic report on biopsy later was hemangioma		
115023				B	130% S	7.2	10,300		5/23/38	500 r—30 r daily	94.9	Improved	3 yr.	
				A		5.4	7,000							
				B		7.9			11/28/38	700 r—30 r daily; discontinued because of leukopenia				
				D			1,800							
				A		6.4	7,100							
				B		7.2			10/27/41	400 r—25 r daily; discontinued (leukopenia)		General condition good		General condition continues good
9	M	55	Gangrene lt. 3th toe; plethora	B	14.9 gm.	6.9	11,000		10/31/38	1000 r—30 r daily	98.0	Toe amputated	3 yr.	Has been well, without further thrombosis
142886				A		4.8	11,600							
						4.5	12,200		12/7/41					
10	F	68	Known polycythemia with previous hemorrhage after minor operations; lesion on vulva at entry	B	128% S	8.9	21,000	Enlarged	2/16/39 2/23/39 4/17/39	200 r—30 r daily; stopped—hemorrhage from biopsy wound		Had been treated with phenylhydrazine; biopsy (vulvectomy): kraurosis		
175527				D		4.2								
				B	18.3 gm.	7.2	17,400	Slight enlargement	12/ 4/39	500 r—25 r daily			2 yr.	
				A	18.1 gm.	6.9	15,500							
						4.7	8,000		7/—/41			Cerebral accident with paralysis; venesections		Bedridden since July, 1941
11	F	65	Dyspnea; varicose veins; phlebitis; ankle edema; plethora	B	20.8 gm.	7.7	15,600	Enlarged	10/ 9/39	650 r—30 r daily	95.0	Much improved	2 yr.	
16700				A	19.5 gm.	6.5	5,200							
						4.1			10/—/40					Patient continues to feel well
					15.1 gm.	5.8	12,500		11/29/41					
12	F	61	Dyspnea; weakness; active duodenal ulcer	B	168% S	7.1	5,300		10/23/39	500 r—30 r daily				General condition good
198626				D			4,800						2 yr.	
				A	20.1 gm.	6.2	6,500							
					16.4 gm.	4.4	6,400		1/13/41					
					17.5 gm.	4.6	6,000		10/20/41					

TABLE I—*Continued*

	Case	Sex	Age	Symptoms	Blood Count				Spleen	Date	Treatment	Oxygen Saturation per cent	Remarks	Length of Remission	Final Status
						Hg	Erythrocytes (million)	Leukocytes							
215738	13	M	47	Hemoptysis 15 yr.; plethora	B	132%	8.5	20,400	Enlarged	11/ 6/39	250 r—30 r daily	91.0 95.0	Sternal biopsy unsatisfactroy	None	
					A	21.8 gm.	9.2	12,700		12/ 4/39 12/21/39	350 r—25 r daily			None	
					B A	16.9 gm.	8.6 5.8	8,400		2/ 6/40 6/—/40	400 r—50 r daily; not completed		Lack of cooperation		
						24.3 gm.	8.1			9/—/40			Venesection—500 cc.		
							8.5			1941			Same condition. Refused further treatment of any sort		Spring, 1942 returned for treatment
226399	14	M	64	Gangrene rt. foot and leg; mental confusion and aphasia 5 mo.	B A	128% S 12.7 gm.	7.7 4.4	14,400 6,100	Enlarged	12/18/39	500 r—25 r daily		Leg amputated; mental condition cleared	2 yr.	
							5.0 5.0			1940 Dec. 1941					Remained relatively normal; feeling well
253145	15	M	53	Peptic ulcer for years; coronary thrombosis 9 mo. before entry; plethora	B A	17.5 gm.	8.3 6.0 5.2	18,500	Enlarged	7/22/40 1/—/41 10/—/41	250 r—30 r daily; not completed; (became blood donor)		Anginal pain persists		Angina persists; no longer acting as blood donor
270452	16	F	37	Known polycythemia over 6 yr.						1934 to 1940	Phenylhydrazine and localized irradiation to spleen and long bones		Nausea and vomiting from drug. Treatment caused temporary falls in red count		
				Migraine attacks	B A	21.4 gm.	7.5 3.3	10,000 4,400	Enlarged Smaller	12/ 9/40	500 r— 25r daily		Some dizziness; generally better	1 yr.	
						20.7 gm.	6.0	5,700		12/22/41					Still some vertigo; improved
287260	17	F	57	Vertigo; blurred vision; paresthesia extremities; nausea, vomiting	B A	22.5 gm.	7.4 4.9	10,000 5,100		2/13/41	500 r—25 r daily	95.0	Symptoms of 2 yr. duration Lowest leukocyte count noted 4,300		
						14.9 gm.	5.1	7,200		11/17/41				8 mo.	In good condition
206930	18	M	40	Headache 3 yr.; lethargy; anorexia; peptic ulcer	B A	17.5 gm. 14.5 gm.	10.5 4.9	12,000 4,600	Enlarged	6/23/41 9/—/41	500 r—20 r daily	89.0		5 mo.	Has remained well
32408	19	F	74	Gangrene tip rt. index finger	B	90%	10.5	22,000		10/20/41	150 r—30 r daily		Had had several venesections (unknown amounts)		Was discharged to home. Nurse called to bring her to hospital; found her dead. Cause unknown
12642	20	M	76	Headaches; vertigo; diabetes	B A	24.0 gm. 11.0 gm.	9.0 4.7	17,000 6,000		6/ 9/41	320 r—20 r daily		Improved in general health	6 mo.	Remains in good condition

CONCLUSIONS

A study of 20 cases of polycythemia vera treated at the Massachusetts General Hospital during the past ten years with small doses of roentgen radiation to large areas of the body has emphasized two important advantages of this method of treatment: first, it caused no definite ill effects; second, it resulted in longer remissions than had followed other forms of therapy. The present technique of treatment is the most satisfactory which has been developed.

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FEBRUARY, 1944

THE AMERICAN JOURNAL OF ROENTGENOLOGY AND RADIUM THERAPY

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Twenty-eighth Annual Meeting: 1944, to be announced.

EDITORIALS

PERFORATED PEPTIC ULCER

IN 1942 there was editorial comment[1] in this JOURNAL on the "Effect of heavy air raids on the incidence of perforated peptic ulcer." This editorial was based on a report of Stewart and Winser. In order to confirm their impressions that there was a definite rise in the incidence of perforated peptic ulcer during air raids, they[2] have again analyzed statistics dealing with this problem from the same group of hospitals and they have come to the conclusion that the increase in perforated peptic ulcer during the period of heavy raiding was statistically significant when compared with the previous three, and the following two years. They have statistical evidence which seems to prove or certainly to favor some general tendency having operated during the air raid period to cause perforations of peptic ulcer and definitely in the two years following the general blitz period there has been a steady decline in the number of perforated peptic ulcers entering the hospitals.

Recently Raw[3] has analyzed a series of 312 cases of perforated gastric and duodenal ulcer and in a discussion of his cases he points out the fact that perforated ulcer should be regarded as one of the most important of abdominal diseases, and that in the particular area in which his statistics were gathered the total number of deaths is considerably greater than the total for appendicitis, though the latter condition is more common. As might be expected, the patients are usually in the prime of life and the fatality rate varies greatly with early diagnosis and the experience of the surgeon in handling such cases. Raw describes the clinical picture which such cases present at the time of perforation and also calls attention to the aid which the roentgen ray can render in such cases. As has been pointed out by a number of observers, the fatality rate runs closely parallel to the number of hours between perforation and operation, and the mortality rate increases with the more advanced age of the individual. While perforations in women are fairly uncommon in his group of cases, as in other statistical studies, it is in women that the diagnosis is so often not made early because in many of these cases there is a mistaken diagnosis of gallstones and the patient's operation is delayed and consequently the probability of a cure from operative procedures is lessened.

In Raw's series of 312 perforated gastric and duodenal ulcers there was a gross mortality of 14.4 per cent and the two most important factors influencing this operative mortality were the age of the patient and the interval before operation.

Since the operation for perforated peptic ulcer is fraught with considerable initial mortality rate and a rather high morbidity rate, it is important to know some of the late results of perforated peptic ulcer. Since the subject has assumed such great importance, particularly in the zones of combat and the cities where bombing has been carried on to a certain great extent, it is of interest to learn the late end-results in a group of cases covering a period of ten years. Such a group of cases was analyzed by Wakeley[4] at the Royal Naval Hospital, Haslar, and a ten year period was chosen

[1] Editorial. Effect of heavy air raids on the incidence of perforated peptic ulcer. AM. J. ROENTGENOL. & RAD. THERAPY, 1942, *47*, 626–627.

[2] Spicer, C. C., Stewart, D. N., and Winser, D. M. de R. Perforated peptic ulcer during the period of heavy air-raids. *Lancet*, Jan. 1, 1944, *1*, 14–15.

[3] Raw, S. C. Perforation of gastric and duodenal ulcers. *Lancet*, Jan. 1, 1944, *1*, 12–14.

[4] Wakeley, C. P. G. Late results of perforated peptic ulcer. *Lancet*, Jan. 1, 1944, *1*, 11–12.

from 1924 to 1934. There were 103 cases admitted to the hospital during this time and all were submitted to operation, with a case fatality of 8 per cent. This is a remarkable figure in contrast with some of the higher mortality rates reported from general civilian hospitals. Part of this low mortality rate is due no doubt to the fact that the average age of the patients was considerably lower than that encountered in civilian practice. The average age of the patients at the Royal Naval Hospital was thirty-two years and, as might be supposed, the men were in a fairly fit condition apart from the fact that they had peptic ulcer. Wakeley attributes another factor to the lower mortality rate, namely the short interval between the perforation of the peptic ulcer and operative interference, the average time being four hours.

In contrast to this group of cases that were cared for in the Royal Naval Hospital, Wakeley collected 102 cases of his own, both hospital and private patients, over the years 1924 to 1934. The average age in this group was forty-six years and the operative mortality rate was 20 per cent. This increase in the mortality rate in the latter group of cases he attributes to the poor condition of the patients themselves and the long period which elapsed between the actual perforation of the ulcer and the time of operation, an average of ten hours.

In the follow up of the group of cases which were operated upon at the Royal Naval Hospital only 5 per cent of the group of cases were untraced. It is of interest in passing that in the Service cases 54 per cent and in the civilian cases 51 per cent of the perforations were gastric in origin.

One interesting and important feature of Wakeley's investigation of the Royal Naval Hospital cases was the fact that 44 per cent of the perforated peptic ulcer cases are still serving in the Royal Navy, the longest time since operation being nearly twenty years and the shortest time nine years. He deduces from a study of his cases in the Royal Naval Hospital that the perforated peptic ulcer patient should not be invalided from the Service but given a careful convalescence, as he is likely to continue to be a useful member of the Royal Navy.

Wakeley also points out the fact that the prognosis is better in perforated cases than in any other form of peptic ulcer. In his group of patients the recurrence of pain or other symptoms was unusual and the patients have lived an ordinary life.

From the analysis of his cases Wakeley concludes that the tendency to reject all men who have had a perforated peptic ulcer is quite unwarranted considering that a high proportion have never had a sign or symptom of their former trouble since operation. In the Royal Navy today, he says, it is quite uncommon to lose a case of perforated peptic ulcer and the operative mortality is about 1 per cent.

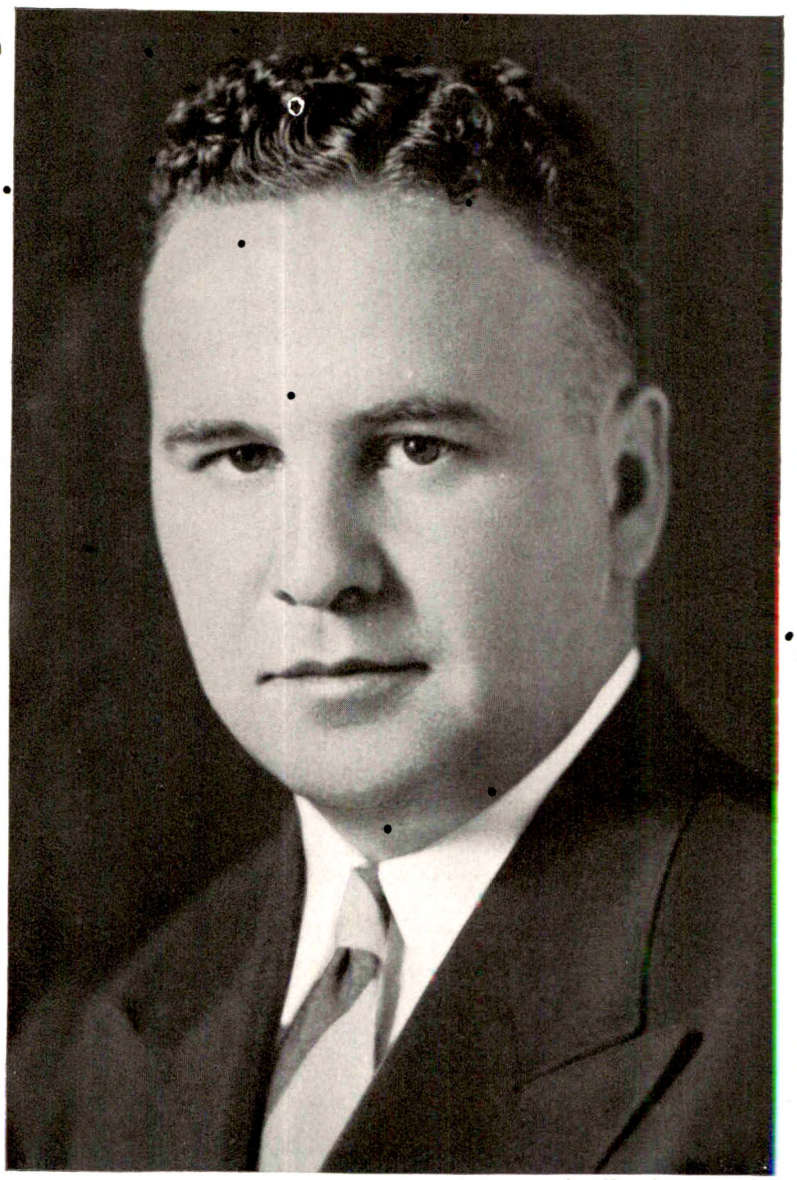
Another important feature in his group of cases is that the incidence of subphrenic abscess following perforated peptic ulcer is reduced to the vanishing point.

It may readily be seen that given a group of cases where a correct diagnosis is made and the proper handling of the patient is then carried out with a high degree of efficiency, the mortality rate is greatly reduced. It only remains for an improvement to be made in the diagnostic side of the problem. Certainly the roentgen ray can, when properly employed, play an important part in the diagnosis of perforated peptic ulcer and those not familiar with the various methods of examination by this means should acquaint themselves with a working knowledge of all the various suggestions which have been advanced for the proper examination of the patient with suspected perforated peptic ulcer.

CUMULATIVE INDEX OF *RADIOLOGY*

THE Radiological Society of North America has just issued a Cumulative Index of their official publication, *Radiology*, covering the years 1923 to 1942, Volumes 1 to 39. In the foreword Doub says that "for a number of years there has been a laudable desire to publish a cumulative index of all the material contained in *Radiology* so that it might be more readily available for reference." The final publication of this Index now in reality makes available the enormous amount of literature which has appeared in the thirty-nine volumes of *Radiology* on radiological and allied subjects. This Cumulative Index fills a great need because *Radiology* has always occupied a position in the forefront of radiological publications and it will prove of immense value to radiologists and medical men the world over. In the organization of the Index the editorial staff has followed the same arrangement of subjects and authors as the *Quarterly Cumulative Index Medicus*. The Index is attractive in its general appearance, style of type and printing.

The publication of the Cumulative Index of *Radiology* is a happy conclusion of a plan which was formulated several years ago. The editorial staff and the collaborators in the work of publishing this Index are to be congratulated on the completion of an arduous task in such a fine manner.



Apex Newsphotos, Fort Worth

L. W. BAIRD

1907-1943

CAPTAIN L. W. BAIRD died of coronary sclerosis on October 6, 1943, at the Station Hospital in Camp Carson, Colorado Springs, Colorado. He was born April 19, 1907, in Edwardsville, Illinois, the son of Henry N. Baird and Maude White Baird. He graduated from the Edwardsville High School, and obtained his M.D. degree from the University of Illinois in 1932. He spent one year as intern at the Santa Clara County Hospital, also one year as resident at this hospital in San Jose, California. He had a three year Fellowship in Radiology at the Mayo Clinic, and obtained a degree of Master of Science in Radiology from the Graduate School of Minnesota. He left the

Mayo Clinic in December, 1936, and was radiologist at the Scott & White Clinic and the Santa Fe Hospital in Temple, Texas, from then until he entered the Army as a Captain in the Medical Corps in November, 1942. He was stationed at Camp Carson as head of the Department of Radiology from then until the time of his death.

Dr. Baird was a member of the Bell County Medical Society, the Texas State Medical Society, and the Radiological Society of North America. He was a Diplomate of the American Board of Radiology, and a member of the American College of Radiology. He also served as Secretary of the Texas Radiological Society from 1941 to 1942. He was a member of the Phi Beta Pi Medical Fraternity and belonged to the First Presbyterian Church in Temple, Texas. He was married to Helen Kestrer on March 31, 1934 in San Jose, California. He is survived by his wife and one son, Robert White Baird. His mother, Mrs. H. N. Baird of Edwardsville, Illinois, and a sister, Mrs. Margarete Whiteside of Fort Worth, Texas, also survive.

Dr. Baird had written scientific papers and appeared on local, state and national programs. He conducted a refresher course on "Carcinoma of the Colon" for members of the Radiological Society of North America at their annual meeting in California in December, 1941.

C. A. Stevenson

SOCIETY PROCEEDINGS, CORRESPONDENCE AND NEWS ITEMS

Items for this section solicited promptly after the events to which they refer.

MEETINGS OF ROENTGEN SOCIETIES*

UNITED STATES OF AMERICA

AMERICAN ROENTGEN RAY SOCIETY
Secretary, Dr. H. Dabney Kerr, University Hospital, Iowa City, Iowa. Annual meeting: To be announced.

AMERICAN COLLEGE OF RADIOLOGY
Secretary, Mac F. Cahal, 540 N. Michigan Ave., Chicago, Ill.

SECTION ON RADIOLOGY, AMERICAN MEDICAL ASSOCIATION
Secretary, Dr. J. T. Murphy, 421 Michigan St., Toledo, Ohio. Annual meeting: 1944, to be announced.

ARKANSAS RADIOLOGICAL SOCIETY
Secretary, Dr. J. S. Wilson, Mack Wilson Hospital, Monticello, Ark. Meets every three months and also at time and place of State Medical Association.

RADIOLOGICAL SOCIETY OF NORTH AMERICA
Secretary, Dr. D. S. Childs, 607 Medical Arts Bldg., Syracuse, N. Y. Annual meeting: To be announced.

RADIOLOGICAL SECTION, BALTIMORE MEDICAL SOCIETY
Secretary, Dr. Walter L. Kilby, Baltimore. Meets third Tuesday each month, September to May.

SECTION ON RADIOLOGY, CALIFORNIA MEDICAL ASSOCIATION
Secretary, Dr. Earl R. Miller, University of California Hospital, San Francisco, Calif.

RADIOLOGICAL SECTION, CONNECTICUT MEDICAL SOCIETY
Secretary, Dr. Max Climan, 242 Trumbull St., Hartford, Conn. Meets bi-monthly on second Thursday, at place selected by Secretary. Annual meeting in May.

SECTION ON RADIOLOGY, ILLINOIS STATE MEDICAL SOCIETY
Secretary, Dr. H. W. Ackemann, 321 W. State St., Rockford, Ill.

RADIOLOGICAL SECTION, LOS ANGELES COUNTY MEDICAL ASSOCIATION
Secretary, Dr. Roy W. Johnson, 1407 S. Hope St., Los Angeles, Calif. Meets on second Wednesday of each month at the County Society Building.

RADIOLOGICAL SECTION, SOUTHERN MEDICAL ASSOCIATION
Secretary, Dr. Roy G. Giles, Temple, Texas.

BROOKLYN ROENTGEN RAY SOCIETY
Secretary, Dr. Leo Harrington, 880 Ocean Ave., Brooklyn, N.Y. Meets monthly on fourth Tuesday, October to April.

BUFFALO RADIOLOGICAL SOCIETY
Secretary, Dr. Joseph S. Gian-Francheschi, 610 Niagara St., Buffalo, N. Y. Meets second Monday of each month except during summer months.

CHICAGO ROENTGEN SOCIETY
Secretary, Dr. Warren W. Furey, 6844 S. Oglesby Ave., Chicago, Ill. Meets second Thursday of each month October to April inclusive at the Palmer House.

CINCINNATI RADIOLOGICAL SOCIETY
Secretary, Dr. Samuel Brown, 707 Race St., Cincinnati, Ohio. Meets third Tuesday of each month, October to May, inclusive.

CLEVELAND RADIOLOGICAL SOCIETY
Secretary, Dr. D. D. Brannan, 11311 Shaker Blvd., Cleveland 4, Ohio. Meets at 6:30 P.M. at Allerton Hotel on fourth Monday each month, October to April, inclusive.

DALLAS-FORT WORTH ROENTGEN STUDY CLUB
Secretary, Dr. X. R. Hyde, Medical Arts Bldg., Fort Worth, Texas. Meetings held in Dallas on odd months and in Fort Worth on even months, on third Monday, at 7:30 P.M.

DENVER RADIOLOGICAL CLUB
Secretary, Dr. Edward J. Meister, 366 Metropolitan Bldg., Denver, Colo. Meets third Friday of each month at Denver Athletic Club.

DETROIT ROENTGEN RAY AND RADIUM SOCIETY
Secretary, Dr. E. R. Witwer, Harper Hospital. Meets monthly on first Thursday from October to May, at Wayne County Medical Society Building.

FLORIDA RADIOLOGICAL SOCIETY
Acting Secretary, Dr. Walter A. Weed, 204 Exchange Bldg., Orlando, Fla. Meetings in May and November.

GEORGIA RADIOLOGICAL SOCIETY
Secretary, Dr. James J. Clark, 478 Peachtree St., Atlanta, Ga. Meets in November and at annual meeting of Medical Association of Georgia in the spring.

RADIOLOGICAL SOCIETY OF KANSAS CITY
Secretary, Dr. Arthur B. Smith, 800 Argyle Bldg., Kansas City, Mo. Meets third Thursday of each month at a place designated by the president.

ILLINOIS RADIOLOGICAL SOCIETY
Secretary, Dr. Wm. DeHollander, St. John's Hospital, Springfield, Ill. Meets three times a year.

INDIANA ROENTGEN SOCIETY
Secretary, Dr. H. C. Ochsner, Methodist Hospital, Indianapolis. Meeting held the second Sunday in May annually.

KENTUCKY RADIOLOGICAL SOCIETY
Secretary, Dr. W. C. Martin, 321 W. Broadway, Louisville. Meets annually in Louisville on first Saturday in Apr.

LONG ISLAND RADIOLOGICAL SOCIETY
Secretary, Dr. Marcus Wiener, 1430-48th St., Brooklyn, N. Y. Meets Kings County Med. Soc. Bldg. monthly on fourth Thursday, October to May, 8:30 P.M.

LOUISIANA RADIOLOGICAL SOCIETY
Secretary, Dr. J. R. Anderson, 1130 Louisiana Ave., Shreveport. Meets annually during Louisiana State Medical Society Meeting.

MICHIGAN ASSOCIATION OF ROENTGENOLOGISTS
Secretary, Dr. E. M. Shebesta, 1429 David Whitney Bldg., Detroit. Three meetings a year, Fall, Winter, Spring.

MILWAUKEE ROENTGEN RAY SOCIETY
Secretary, Dr. C. A. H. Fortier, 231 W. Wisconsin Ave., Milwaukee, Wis. Meets monthly on second Monday at University Club.

MINNESOTA RADIOLOGICAL SOCIETY
Secretary, Dr. Annette T. Stenstrom, 1218 Medical Arts Bldg., Minneapolis, Minn. One meeting a year at time of Minnesota State Medical Association.

NEBRASKA RADIOLOGICAL SOCIETY
Secretary, Dr. D. A. Dewell, Medical Arts Bldg., Omaha, Nebr. Meets third Wednesday of each month, at 6 P.M. at either Omaha or Lincoln.

NEW ENGLAND ROENTGEN RAY SOCIETY
Secretary, Dr. Hugh F. Hare, Lahey Clinic, Boston, Mass. Meets monthly on third Friday, Boston Medical Library.

RADIOLOGICAL SOCIETY OF NEW JERSEY
Secretary, Dr. H. J. Perlberg, 921 Bergen Ave., Jersey City. Meets annually at time and place of State Medical Society. Mid-year meetings at place chosen by president.

NEW YORK ROENTGEN SOCIETY
Secretary, Dr. Ramsay Spillman, 115 East 61st St., New York City. Meets monthly on third Monday, New York Academy of Medicine, at 8:00 P.M.

NORTH CAROLINA ROENTGEN RAY SOCIETY
Secretary, Dr. Major Fleming, Rocky Mount, N. C. Annual meeting at time and place of State Medical Society. Mid-year scientific meeting at place designated.

NORTH DAKOTA RADIOLOGICAL SOCIETY
Secretary, Dr. L. A. Nash, St. John's Hospital, Fargo. Meetings held by announcement.

* Secretaries of Societies not here listed are requested to send the necessary information to the Editor.

Central New York Roentgen Ray Society
Secretary, Dr. C. F. Potter, 820 S. Crouse Ave., Syracuse. Three meetings a year. January, May, November.

Ohio Radiological Society
Secretary, Dr. J. E. McCarthy, 707 Race St., Cincinnati. Meets at time and place of annual meeting of Ohio State Medical Association.

Pacific Roentgen Society
Secretary, Dr. L. H. Garland, 450 Sutter St., San Francisco, Calif. Meets annually, during meeting of California Medical Association.

Pennsylvania Radiological Society
Secretary, Dr. L. E. Wurster, 416 Pine St., Williamsport.

Philadelphia Roentgen Ray Society
Secretary, Dr. R. P. Barden, University Hospital, Meetings first Thursday of each month from October to May inclusive at 8:15 P.M., in Thompson Hall, College of Physicians, 19 S. 22d St.

Pittsburgh Roentgen Society
Secretary, Dr. R. G. Alley, 4800 Friendship Ave. Meets second Wednesday each month, 4:30 P.M., October to June, Pittsburgh Academy of Medicine.

Rochester Roentgen Ray Society, Rochester, N. Y.
Secretary, Dr. Sidney Larson, Strong Memorial Hospital. Meets monthly on third Monday from October to May, inclusive, 8 P.M. at Strong Memorial Hospital.

Rocky Mountain Radiological Society
Secretary, Dr. A. M. Popma, 220 N. First St., Boise, Idaho.

St. Louis Society of Radiologists
Secretary, Dr. P. C. Schnoebelen, 462 N. Taylor Ave. Meets fourth Wednesday of each month, except June, July, August, and September, at a place designated by the president.

San Diego Roentgen Society
Secretary, Dr. Henry L. Jaffe, Naval Hospital, Balboa Park, San Diego, Calif. Meets monthly on first Wednesday at dinner.

San Francisco Radiological Society
Secretary, Dr. Martha Mottram, 450 Sutter St., San Francisco. Meets monthly on third Thursday at 7:45 P.M., first six months of year at Toland Hall, University of California Hospital, second six months at Lane Hall, Stanford University Hospital.

Shreveport Radiological Club
Secretary, Dr. R. W. Cooper, Charity Hospital, Shreveport, La. Meets monthly on third Wednesday, at 7:30 P.M., September to May inclusive.

South Carolina X-Ray Society
Secretary, Dr. T. A. Pitts, Baptist Hospital, Columbia, S. C. Meets in Charleston on first Thursday in November, also at the time and place of South Carolina State Medical Association.

Tennessee Radiological Society
Secretary, Dr. J. M. Frère, 707 Walnut St., Chattanooga, Tenn. Meets annually at the time and place of the Tennessee State Medical Association.

Texas Radiological Society
Secretary, Dr. Herman Klapproth, Sherman, Texas.

University of Michigan Department of Roentgenology Staff Meeting
Meets each Monday evening from September to June, at 7 P.M. at University Hospital.

University of Wisconsin Radiological Conference
Secretary, Dr. E. A. Pohle, 1300 University Ave., Madison, Wis. Meets every Thursday from 4:00–5:00 P.M., Room 301, Service Memorial Institute.

Virginia Radiological Society
Secretary, Dr. E. L. Flanagan, 116 E. Franklin St., Richmond, Va. Meets annually in October.

Washington State Radiological Society
Secretary, Dr. Thomas Carlile, 1115 Terry St., Seattle. Meets fourth Monday each month, October through May, College Club, Seattle.

X-ray Study Club of San Francisco
Secretary, Dr. J. M. Robinson, University of California Hospital. Meets monthly in evening on third Thursday.

Cuba

Sociedad de Radiología y Fisioterapia de Cuba
President, Dr. J. Manuel Viamonte, Hospital Mercedes, Habana, Cuba. Meets monthly in Habana.

British Empire

British Institute of Radiology Incorporated with the Röntgen Society
Medical Members' meeting held monthly on third Friday at 2:30 P.M. and Ordinary Meeting at same time on following Saturday, October to May, 32 Welbeck St., London, W.1.

Section of Radiology of the Royal Society of Medicine (Confined to Medical Members)
Meets on the third Friday of each month at 4:45 P.M. at the Royal Society of Medicine 1, Wimpole St. London, W. 1.

Faculty of Radiologists
Secretary, Dr. M. H. Jupe, 32 Welbeck St., London, W. 1 England.

Section of Radiology and Medical Electricity, Australasian Medical Congress
Secretary, Dr. H. M. Cutler, 130 Macquarie St., Sydney, New South Wales.

Radiological Section of the Victorian Branch of the British Medical Association
Secretary, Dr. Keith Hallam, St. George's Hospital, K.E.W., Melbourne, E. 4, Victoria, Australia. Meets monthly from March to November inclusive.

Canadian Association of Radiologists
Secretary, Dr. A. D. Irvine, 540 Tegler Bldg., Edmonton, Alberta.

Section of Radiology, Canadian Medical Association
Secretary, Dr. C. M. Jones, Ingles St., Ext. Halifax N. S.

Radiological Section, New Zealand British Medical Association
Secretary, Dr. Colin Anderson, Invercargill, New Zealand. Meets annually.

South America

Sociedad Argentina de Radiología
Secretary, Dr. Guido Gotta, Buenos Aires, Argentina. Meetings are held monthly.

Continental Europe

Sociedad Espanola de Radiología y Electrología
Secretary, Dr. J. Martin-Crespo, Fuencarral 7. Madrid, Spain. Meets monthly in Madrid.

Société Suisse de Radiologie (Schweizerische Röntgen-Gesellschaft)
Secretary for French language, Dr. A. Grosjean La Chaux de Fonds.
Secretary for German language, Dr. Scheurer, Molzgasse Biel. Meets annually in different cities.

Societatea Romana de Radiologie si Electrologie
Secretary, Dr. Oscar Meller, Str. Banul Măcăcine 30, S. I., Bucuresti, Roumania. Meets second Monday in every month with the exception of July and August.

All-Russian Roentgen Ray Association, Leningrad: USSR in the State Institute of Roentgenology and Radiology, 6 Roentgen St.
Secretaries, Drs. S. A. Reinberg and S. G. Simonson. Meets annually.

Leningrad Roentgen Ray Society
Secretaries, Drs. S. G. Simonson and G. A. Gusserin. Meets monthly, first Monday at 8 o clock, State Institute of Roentgenology and Radiology, Leningrad.

Moscow Roentgen Ray Society
Secretaries, Drs. L. L. Holst, A. W. Ssamygin and S. T. Konobejevsky. Meets monthly, first Monday, 8 P.M.

Scandinavian Roentgen Societies
The Scandinavian roentgen societies have formed a joint association called the Northern Association for Medical Radiology, meeting every second year in the different countries belonging to the Association.

NEW OFFICERS

The following officers of the American Roentgen Ray Society were recently elected for the year 1943–1944. The vote of the Society's members was taken by mail since the annual meeting had been postponed because of the World War. *President:* Dr. Sherwood Moore, St. Louis, Missouri; *President-Elect:* Dr. Lyell C. Kinney, San Diego, California; *First Vice-President:* Dr. Paul C. Hodges, Chicago, Illinois; *Second Vice-President:* Dr. Aubrey O. Hampton, Boston, Massachusetts; *Secretary:* Dr. H. Dabney Kerr, Iowa City, Iowa; *Treasurer:* Dr. J. Bennett Edwards, Leonia, New Jersey; *Historian:* Dr. Ramsey Spillman, New York, New York.

RADIOLOGICAL SECTION, LOS ANGELES COUNTY MEDICAL ASSOCIATION

At the meeting of the Radiological Section of the Los Angeles County Medical Association, held on December 8, 1943, the following officers were elected for the ensuing year: Dr. Benjamin H. Sherman, *President;* Dr Donald R. Laing, *Vice-President;* Dr. Roy W. Johnson, *Secretary;* Dr. Henry Snure, *Treasurer.*

SAN DIEGO ROENTGEN SOCIETY

Through the efforts of Dr. Keener, Senior Medical Officer in Roentgenology at the Naval Hospital, San Diego, California, a group of radiologists met during the latter part of 1943 and organized the San Diego Roentgen Society to include the local radiologists and those who are stationed in the vicinity in the various Services. The group will meet on the first Wednesday of each month for dinner to be followed by a case seminar. Dr. Henry L. Jaffe of Chicago was elected Secretary. Dr. Jaffe is stationed at the Naval Hospital, Balboa Park, San Diego.

DIRECTORY OF MEDICAL SPECIALISTS

Announcement is made that the Directory of Medical Specialists is now to be published by the A. N. Marquis Company of Chicago, publishers of "Who's Who in America." Previous editions have been published for the Advisory Board for Medical Specialties by the Columbia University Press of New York City.

It is planned not to issue the next edition before 1945, on account of the war, but the A. N. Marquis Company will publish a supplemental list of all those who have been certified by the American Boards since the last (1942) edition of the Directory, totaling about 3,600. This is to be distributed at cost, and monthly or bimonthly bulletins listing successful candidates for certification at examinations during the additional interim before the next edition are to be issued as a subscribers' service.

Dr. Paul Titus (Pittsburgh) of the American Board of Obstetrics and Gynecology will continue as the Directing Editor, and Dr. J. Stewart Rodman (Philadelphia) of the American Board of Surgery continues as Associate Editor. The Editorial Board will be composed, as before, of the Secretaries of the fifteen American Boards.

Communications should be addressed to the Directing Editor, Directory of Medical Specialists, 919 No. Michigan Avenue, Chicago (11), Illinois.

BOOK REVIEWS

Books sent for review are acknowledged under: Books Received. This must be regarded as a sufficient return for the courtesy of the sender. Selections will be made for review in the interest of our readers as space permits.

OCCUPATIONAL TUMORS AND ALLIED DISEASES. By W. C. Hueper, M. D., Assistant Director and Principal Pathologist, Warner Institute for Therapeutic Research, New York City. Cloth. Price, $8.00. Pp. 896. Springfield, Illinois; Charles C Thomas, 1942.

The author has set down in 860 pages an exhaustive review of the world's literature pertaining to the occupational etiology of tumors of the human body. Neoplasms of the various organs and anatomical systems are considered in order and the scope of the book is indicated by a subject index of 46 pages and many elaborate bibliographies, each containing hundreds of references. In one of the final chapters the relation of occupational neoplasia to the various theories of cancerigenesis is considered at some length.

With so little actually known about the cause of cancer the reviewer finds it difficult to justify the labor involved in the preparation of this massive treatise but the author explains that it is intended not only for physicians but for governmental agencies, legislative bodies, workmen's compensation boards, lawyers, engineers, life insurance executives, biologists, and chemists and for this reason many detailed technical data are included. Occupational hazards are stressed throughout the book and the concluding chapter deals with the medicolegal and public health aspects of the subject.

CHARLES L. MARTIN

CLINICAL ROENTGENOLOGY OF THE CARDIOVASCULAR SYSTEM. By Hugo Roesler, M.D., F.A.C.P., Associate Professor of Roentgenology and Cardiologist, Department of Medicine, Temple University School of Medicine; Cardiologist, Temple University Hospital, Philadelphia. Second Edition. Cloth. Price, $7.50. Pp. 480, with 337 illustrations. Springfield, Illinois: Charles C Thomas, 1943.

The second edition of this authoritative book is even more comprehensive than the first. Adequate abstracts of the newer diagnostic procedures are given for arteriography, venography, Robb-Steinberg opacification of the cardiac chambers and great vessels, cinematography, kymography and for the detection of intracardiac deposits of calcium. The chapter on Cardiac Measurement includes a thorough discussion of various complicated prediction formulae that will be of interest to those familiar with higher mathematics. New and interesting studies are quoted on the influence of athletics, of avitaminosis, and of the metabolic diseases on the heart. The chapters on Cardiovascular Disease from the Viewpoint of Structural Changes, on Diseases of the Aorta, and on Pericarditis are full of practical clinical observations. The use of small numbers to identify the features of the new illustrations makes them much easier to read and is a distinct advantage.

In general the volume has been brought up to date with emphasis rightly placed on the newer roentgenographic procedures in the study and diagnosis of cardiovascular disease. This progressive viewpoint together with the greater use of the contributions of medical men of this country with more references to our accessible literature is a decided improvement and makes the second edition a most worth-while text. It should be read by all those interested in heart and vascular diseases.

WENDELL G. SCOTT

A MANUAL OF RADIOTHERAPY. By Murray M. Friedman, M. D., Assistant Professor of Radiology, College of Physicians and Surgeons, Columbia University; Assistant Radiologist, Presbyterian Hospital, New York. Paper. Price, $1.50. Pp. 86, with 30 illustrations. Ann Arbor, Michigan: Edward's Brothers, Inc., 1942.

This small paper bound manual gives in brief outline form the factors and dosages used in the Department of Radiotherapy at the Presbyterian Hospital in New York. A short description of each lesion is followed by details of the technique recommended and a limited bibliography. The radium dosage tables and the curves of Paterson and Parker and of Quimby are included, and a few pages are devoted to roentgen injuries and irradiation sickness.

The student will find this outline a valuable guide, but its brevity constitutes its main defect. Certainly the beginner should receive added information before undertaking the treatment of patients. Some of the roentgen irradiation techniques lack the boldness needed for the best results, and such important malignant conditions as cancer of the pharynx, cancer of the cheek and large cancers of the skin are not considered at all. The book could also be improved by the addition of a chapter on the physics of roentgen-ray dosage and more detailed information relative to the practical application of the curves and tables covering radium dosage.

CHARLES L. MARTIN

VOL. 51, NO. 2

DEPARTMENT OF TECHNIQUE

Department Editor: ROBERT B. TAFT, M.D., B.S., M.A., 103 Rutledge Ave. Charleston, S. C.

A SIMPLE METHOD FOR THE ACCURATE MEASUREMENT OF INTRATHORACIC SHADOWS

By G. S. PESQUERA, M.D., *and* E. C. LASHER, R.T.
Metropolitan Life Insurance Company Sanatorium, Department of Roentgenology
MOUNT McGREGOR, NEW YORK

THE accurate determination of the location and the size of objects observed as shadows in roentgenograms of the chest has been attempted by many investigators in the field of roentgenology. The structures that have received particular attention have been foreign bodies, cavities, tumors, and the heart. Their approximate size and location have been derived from the use of various roentgenological techniques together with the aid of triangulation and computed formulae. The methods most frequently used for heart measurements have been the transverse diameter of heart shadows from roentgenograms of the chest taken at 6 or 7 feet (target-film distance), from the tracings obtained from the orthodiagram, and of late by the kymograph.

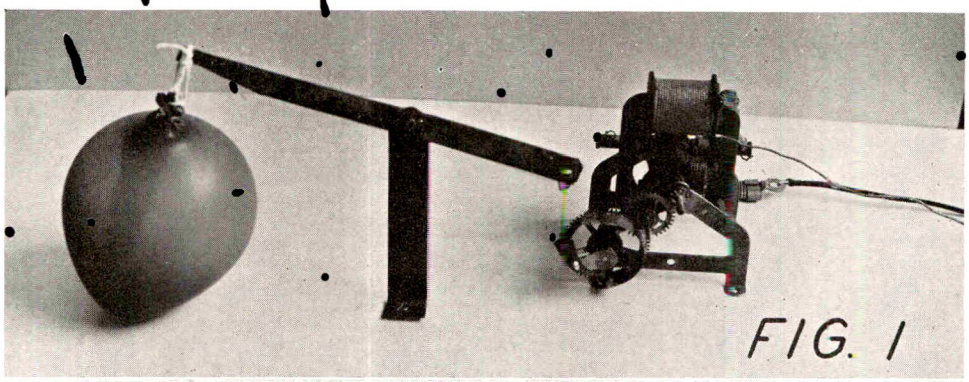
Body section roentgenography is accomplished by making a series of roentgenograms of a subject while the roentgen tube and the film are moved about a fulcrum placed at the level of the section being roentgenographed, the fulcrum being varied for each section taken and varied as many times as there are sections desired. This motion causes gross blurring of all structures except those within the section at the level of the fulcrum. This procedure makes it possible for the roentgenologist to determine with accuracy the distance between the film and the object under consideration. The body section roentgenogram which shows the sharpest image of the object is a roentgenogram of a thin section parallel to and at the same distance from the table top as is the object. Once this factor is determined, the object-film distance is obtained by the addition of the table top to film distance. The difference between the known target-film and the derived object-film distance gives the target-object distance.

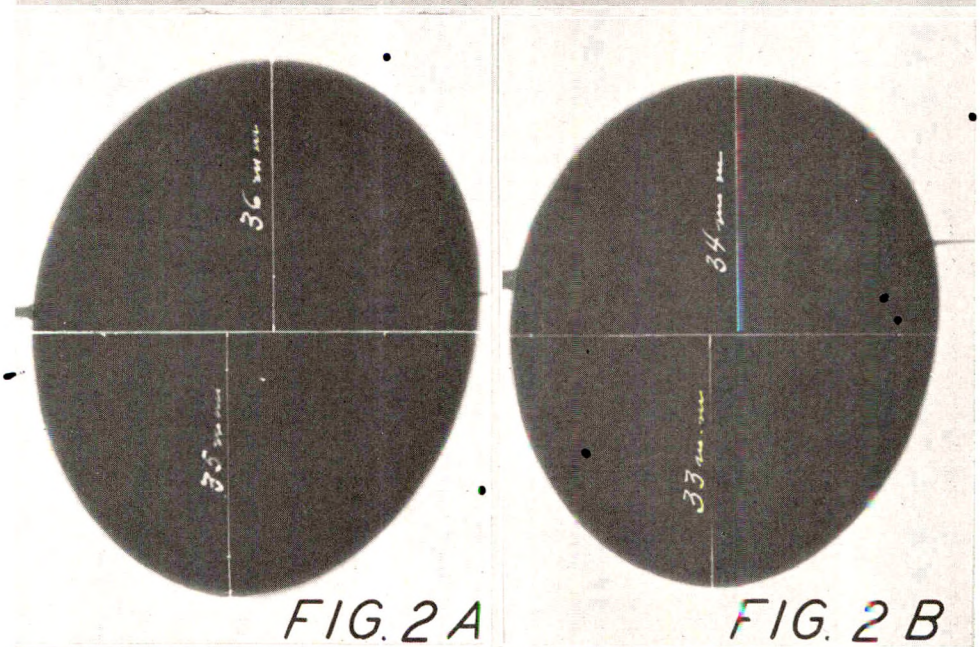
Andrew and Warren have evolved a simple formula for the computation of the actual size of any object being roentgenographed when the target-film distance, the target-object distance and the image size are known:

$$\frac{\text{object-size}}{\text{image-size}} = \frac{\text{target-object distance}}{\text{target-film distance}}$$

We present herewith a simple, and, we believe, an accurate method of measuring objects such as intrapulmonary cavities and foreign bodies, and suggest it as a method for determining heart size. If this method provides a correct answer for object size, then a permanent record obtained at one date may be confidently compared with a record of the same object obtained at any future date. Since the object itself is measured, then such variable factors as gain or loss in weight or changes in posture would no longer need to be considered in the calculation.

We have undertaken to determine the accuracy of measurements with the above method by placing coins of various sizes on blocks at different levels above the roentgen-ray table top. A series of body





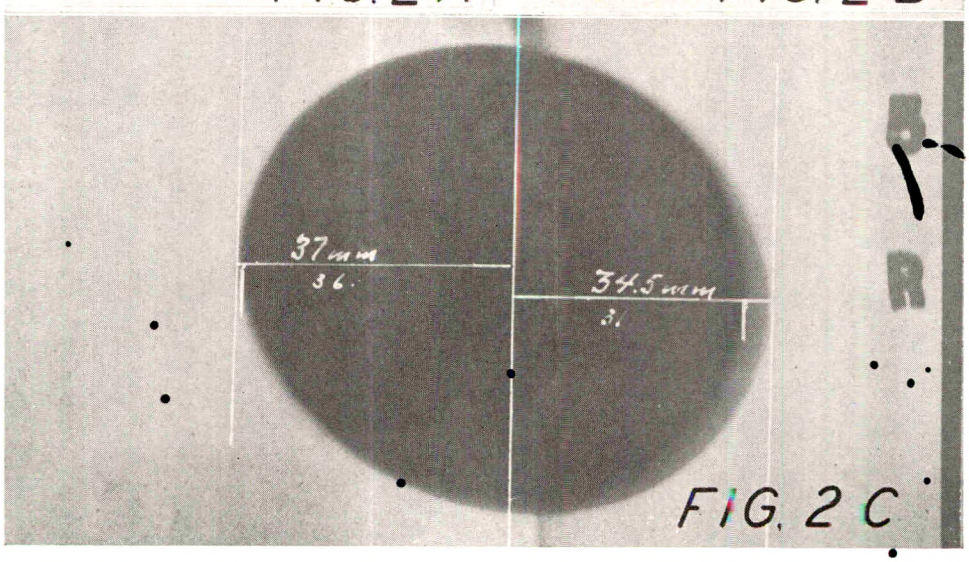


FIG. 1. Model of artificial or simulated heart.

FIG. 2. Body section roentgenograms of heart showing standard cardiac transverse diameter measurement. *A*, diastole, stationary; *B*, systole, stationary; *C*, diastole and systole, heart in motion.

section roentgenograms were then made and from these the one presenting the sharpest image of the coin was selected.

One example will be sufficient to illustrate the technique employed. A five-cent piece measured by micrometer was found to be 2.12 centimeters in diameter. The roentgenogram which gave the sharpest shadow of this coin was taken at 13.5 cm. from the table top. Adding the distance from the table top to the film, usually called Bucky distance when a Potter-Bucky diaphragm is used, we have an object-film distance of 18.5 cm., which when subtracted from the target-film distance of 101.6 cm. gives a target-object distance of 83.1 cm. The image size of the coin measured 2.6 cm. in diameter. Applying the formula of Andrew and Warren we have:

$$\frac{x}{2.6} = \frac{83.1}{101.6}$$

where x equals the object size.

The size of the coin as obtained from this equation was 2.126 cm. which gives an error of 0.006 cm. as compared with the actual measured size of 2.12 cm. The maximal error we encountered in many such calculations was 0.035 cm. We believe that the errors are due to our inability to measure, by the instruments employed, the shadow on the roentgenogram with an accuracy greater than ±0.025 cm.

To simulate a heart in motion a model was made (Fig. 1) consisting of a rubber balloon filled with an opaque solution (potassium iodide) attached to a fixed base and to a movable arm activated by a small electric motor. With the arm in motion, the balloon was alternately stretched and relaxed at the rate of 70 times per minute. When fully stretched the balloon measured 6 cm. in greatest diameter, and when fully relaxed, 6.4 cm. Body section roentgenograms were made with the object stationary at its smallest diameter (systole), and at its largest diameter (diastole), as well as with the simulated heart in motion. The roentgenograms selected for measurement were those showing the largest and sharpest shadows, since we desired the maximum diameter of the object.

For diastole the roentgenogram selected (Fig. 2*A*) was taken at the 4.35 cm. level from the table top. Adding the Bucky distance of 5 cm. we have an object-film distance of 9.35 cm. When this is subtracted from the target-film distance of 101.6 cm. a target-object distance of 92.25 cm. is obtained. The image (heart silhouette) was measured by drawing a central vertical line from which horizontal lines were drawn at right angles to the outermost point of the curve of each semicircle. The measurement obtained was 7.1 cm.

$$\frac{x}{7.1} = \frac{92.25}{101.6}$$

From the formula the size obtained was 6.446 cm. whereas the actual size was 6.4 cm.

The roentgenogram selected for systole (Fig. 2*B*) gave an object-film distance of 9.9 cm. which left a target-object distance of 91.7 cm. The image size was 6.7 cm

$$\frac{x}{6.7} = \frac{91.7}{101.6}$$

The calculated size was found to be 6.047 cm. and the measured size was 6.0 cm.

Body section roentgenograms taken with the model in motion (Fig. 2*C*) gave approximately the same results: Diastole, 6.446 cm.; systole, 6.0405 cm.

SUMMARY

Evidence is submitted to show that the actual size of an object is determinable within narrow limits when body section roentgenography and the simple formula devised by Andrew and Warren are employed. For practical purposes the discrepancy between computed and actual size may be ignored. We have also shown that for practical purposes the measurements of a simulated heart in motion are as accurate as when the object is stationary. We suggest the use of this method when accurate transverse measurements of the heart are desired.

We wish to express our gratitude to Dr. E. M. Medlar of our Research Laboratory for his constructive criticism.

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VOL. 51, NO. 2

AN EASY METHOD OF FOREIGN BODY LOCALIZATION READ DIRECTLY FROM THE FLUORSCOPIC SCREEN*

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THE following is an easy method by which the depth of a foreign body can be read directly during the roentgenoscopic procedure from a scale placed upon the screen.

The mathematical principles of the method are as follows:

This is illustrated in Figure 1. The principle involved is to obtain a certain angle of rays from the focal spot of the tube with a specified distance between two objects, e.g., the skin marker and the foreign body. This can be obtained as follows: In the line of the central ray of the focal spot, place a metallic marker, *M*, over the position of the foreign body, *FB*. Opposite the marker, *M*, place another marker, *M'*, at a certain distance, e.g., $\frac{1}{4}$ inch, on the same horizontal plane. Now shift the tube and screen so that the shadow of the foreign body, *FB*, will be superimposed on the shadow of the second marker, *M'*, on the screen. The distance of this shift, *OB*, from the central line of rays on the screen establishes a relationship between the depth of the foreign body, *FB*, from the markers. The depth of the foreign body, *FB*, from the markers determines the angle at which the rays from the focal spot with which the shadow of *FB* will be superimposed on the shadow *M'*. This can readily be seen from Figure 1. This angle will always be the same no matter what the position of the foreign body, *FB*, and markers, *M* and *M'*, are in relationship to the focal spot and screen, providing the distances between *FBM* and *M'* and *M* are kept constant. This is shown by the broken lines in Figure 1, when the foreign body *FB* and the markers are placed in a different position in reference to the tube and screen and all the other factors are kept constant.

Triangle *X* will be equal to triangle *Y* by construction. From Figure 1 it will also be seen that triangle *X* will be similar to triangle *OBFS* in the broken lines and it will also be similar to triangle *O'B'FS*, as the angles formed by the parallel lines will

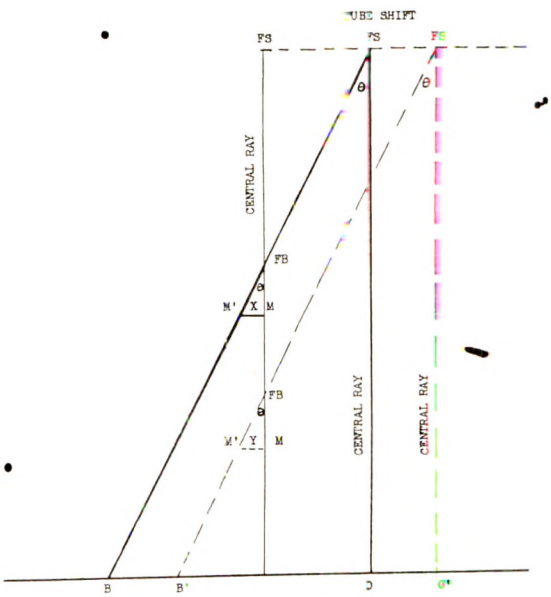


FIG. 1. The markers *M* and *M'* are placed at a certain distance from each other on the same horizontal plane, and at a certain distance from the foreign body *FB*. The figure illustrates that a certain angle θ will be obtained with each distance between the foreign body and *M* and *M'* forming equal and similar triangles. The figures in the broken lines illustrate that if the distance between *FB* and *M* and *M'* is placed in a different position in reference to the focal spot but the distance between them is kept constant, the distance *OB* and *O'B'* will be equal although the tube shift is different.

* Published with permission of the Medical Director, Veterans Administration, who assumes no responsibility for the opinions expressed or conclusions drawn by the author.

be equal. The right angle triangles, $OBFS$ and $O'B'FS$, will be equal, as is evident from the figure, as FSO and FSO', the focal screen distances are the same and parallel. The lines, FSB and FSB', are parallel and equal, and therefore the angles θ will be equal. The distances, OB and $O'B'$, will be equal. The distance of the shift on the screen OB is directly proportional to the angle θ, which is proportional to the distance MFB. Each distance between FB and M and M' will give a certain definite angle θ and hence a relationship is established between the depth of the foreign body and the shift at which the marker M' will be superimposed on the screen. It is also seen from Figure 1 that although the distance of the tube shift will be different if the constant depth of the foreign body and markers are varied in the relationship to the focal spot and screen, the shift at which the shadow of FB and M' will be superimposed will be the same on the screen. It is a simple matter to calculate the shift OB for any depth between the markers and the foreign body.

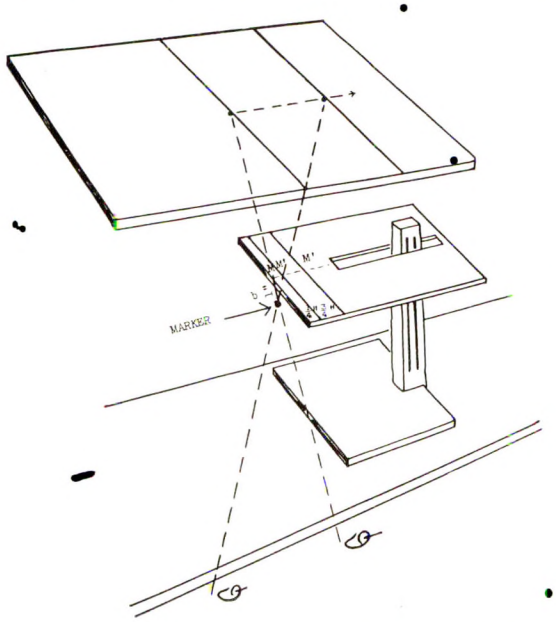


FIG. 2. The apparatus shows how the cross-board with its three wires $\frac{1}{4}$ inch, $\frac{3}{4}$ inch apart with the 1 inch projecting piece (b) can be raised or lowered or moved in a horizontal position over the patient. The wires are used as guides so that their shadows will project on the screen upon which a scale can be placed and read. The end wire is centered with the central line of the screen and with the foreign body and the tube and screen shifted so that the shadow of the foreign body projects on one of the wires.

It follows, since the triangles are similar, that FBM the depth of the foreign body: FSO the tube screen distance $= MM'$, the distance between the markers: OB the shift at which the shadow of M' and FB superimpose on the screen. Taking various depths between the foreign body and marker and substituting them in the above equation and selecting a focal spot screen distance, the shift OB can be calculated mathematically.

PRACTICAL APPLICATION OF THIS PRINCIPLE

A simple apparatus that can be constructed without difficulty by any carpenter, cabinet maker or machinist is used. This consists of a stand (Fig. 2) about 9 inches high and an adjustable wooden

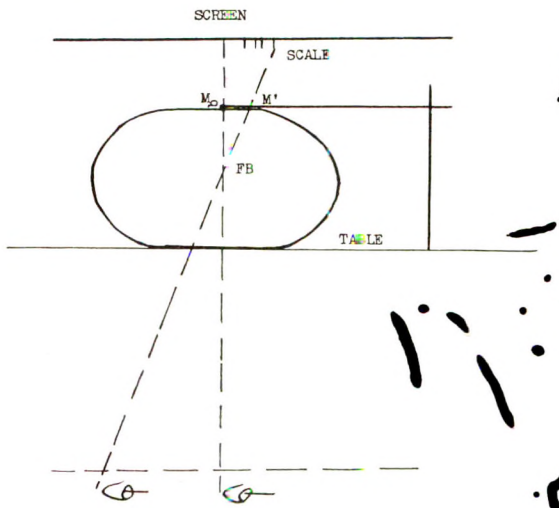
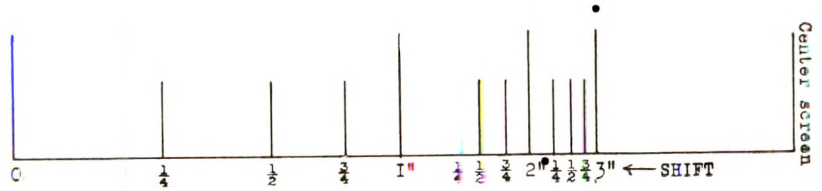


FIG. 3. To illustrate the method and procedure, the cross-bar is lowered so the projecting piece (b) is directly over the foreign body. The shadow of the end wire, the center line of the screen and the foreign body are superimposed and the tube and screen shifted until the shadow of the foreign body is superimposed on that of one of the guide wires and the depth read directly from a scale placed on the screen by the reflected light.

cross-bar about 8 or 9 inches wide that can be raised or lowered or moved in a horizontal direction. Upon the cross bar are placed three wires, $\frac{1}{4}$ inch and $\frac{3}{4}$ inch apart. Over the center of the first wire is placed, on the under surface, a projecting piece (*b*) 1 inch from the top of the first wire. During the localization procedure the cross-bar until the projecting piece (*b*) is directly over and upon the shadow of the needle. Then center the center line of the screen with the shadow of the end wire of the cross-bar and the shadow of the needle, so that these three shadows superimpose (Fig. 2). Now shift the tube and screen so that the shadow of the needle is superimposed on the shadow of the $\frac{1}{4}$ inch distant wire on the screen. From Figure 1 it can be seen that similar triangles are formed and the mathematical relationship below holds. *FBM* (the distance of the foreign body to the skin marker *M*): *FSO* (the focal screen distance) = *MM'* (the distance between the $\frac{1}{4}$ inch wire markers): *OB* (the shift or the screen where the shadows are superimposed). *OB* can be measured directly on the screen and the equation solved for *FSO*, the focal screen distance.



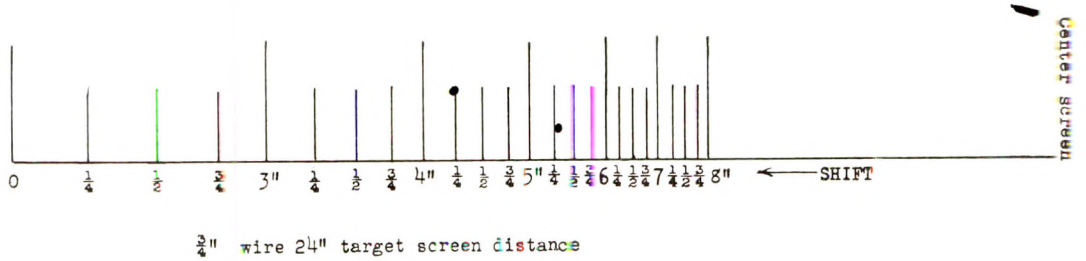


FIG. 4. Two scales are shown, one being the $\frac{1}{4}$ inch guide wire, the other the $\frac{3}{4}$ inch guide wire for a 24 inch target screen distance. By placing the scales on the side from the central line of the screen (Fig. 5) and by using the apparatus at a certain distance the screen is raised or lowered until the scale corresponds to this distance. The scale gives the distance from the end of the projecting piece (*b*) to the foreign body. See text.

cross-bar is so placed that the projecting piece (*b*) is on the patient's skin directly over the foreign body in the central ray of the focal spot.

The next step is to construct a scale. Select a convenient distance between the table top and screen, e.g., 12 inches, and then determine the focal spot screen distance. If this is unknown, a simple method to obtain this is as follows: Take a metallic marker—a needle stuck in a piece of adhesive tape is excellent for this purpose. Take the above apparatus and lower the

To obtain the central line of the focal

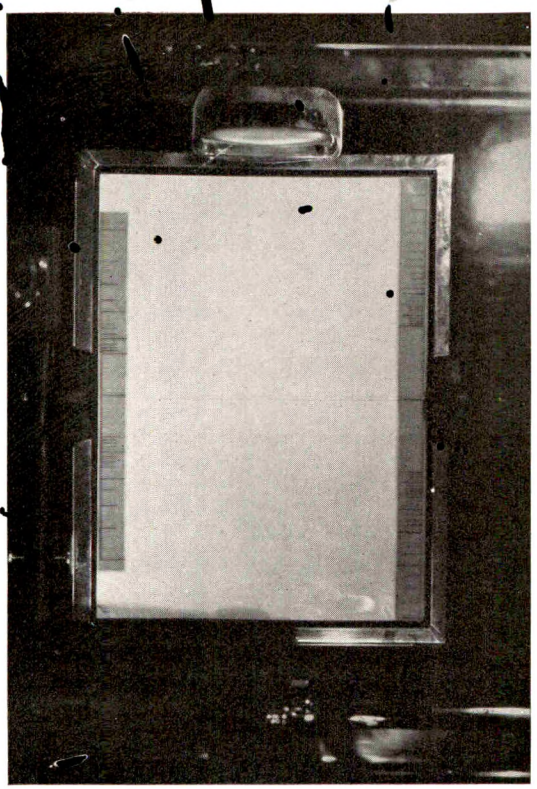


Fig. 5. The scales are placed on either side of the central ray. The scale is read directly during roentgenoscopy by the reflected light from the screen. The scales are for a 24 inch target screen distance, one calculated for $\frac{1}{4}$ inch guide wire, the other for the $\frac{3}{4}$ inch guide wire.

spot place a ruler with a metallic edge at right angles to the table top. Raise and lower the screen and shift the screen until a position is reached where the shadow of the ruler edge is stationary when the screen is raised or lowered. The center line of the screen is not necessarily in the center of the diaphragm openings.

After the focal spot screen distance *FSO* is determined, the shift *OB* for any depth can be calculated from the equation above. Practically this is done in $\frac{1}{4}$ inch steps, as the intermediate positions can be determined by interpolation.

The calculation is done for the first 4 inches using the $\frac{1}{4}$ inch wire guides. For the next 4 inches it is better to use the 1 inch wire guide as the distance of the shift *OB* would become small and hence difficult to measure practically. Hence two scales are used, one using the $\frac{1}{4}$ inch wire, the other the 1 inch wire.

Scales calculated for a 24 inch focal screen distance are illustrated. If desired, these scales can be cut out and transferred to the screen from the central line, as illustrated in Figures 3, 4 and 5. Place the apparatus over a marker, e.g., a needle at a certain distance, e.g., 2 inches from the end of the projecting piece (*b*) to the needle. Center the shadows of the end wire with the projecting piece (*b*) over the central line of the screen and center this over the needle. Then shift the tube and screen until the needle shadow *M* and the $\frac{1}{4}$ inch wire shadow *M'* superimpose. Raise or lower the screen until the superimposed shadows of the needle and $\frac{1}{4}$ inch wire *M'* correspond to 2 inches on the scale. This will give a 24 inch focal spot screen distance. It is useful to measure and cut a wooden block of this distance from the table top, and all that is necessary to obtain the same focal spot screen distance is to bring the screen down until it reaches the top of the wooden block.

The two scales can be transferred to some clear roentgen film using india ink and fixing them to the edge of the screen (Fig. 4 and 5). This will not interfere with subsequent roentgenoscopic procedure and will enable one to read directly the scale during the examination by the reflected rays from the screen. It is also more convenient to duplicate the scales on each side of the central line.

A scale can also be made directly by merely raising the cross-bar at definite distances from the needle and noting the distance of the shifts with a skin marking pencil.

To localize the depth of a foreign body is now a simple matter taking a few minutes and is read directly during the roentgenoscopic examination.

Step 1: Place the patient in the surgical position, e.g., the position at which he would be operated upon. Locate the foreign body and select a certain part of it and

center this with the center line of the screen.

Step 2: Place the apparatus upon the table and lower the cross-bar until the projecting piece (*b*) is upon the skin. Then center the shadow of the foreign body with the shadow of the end wire of the cross-bar with the center line of the screen until they are superimposed.

Step 3: Shift the tube and screen so that the same position of the foreign body chosen will be superimposed on either the shadow of the $\frac{1}{4}$ inch wire or the $\frac{3}{4}$ inch wire, and read the depth directly from the scale on the screen.

It can readily be seen that the shift of the tube and screen can be done either at right angles to the long axis of the tube or parallel to the table. It is only necessary to change the position of the wires in reference to the cross-board and the position of the scales.

The author wishes to express his appreciation to Dr. Foster J. Curtis, Chief Medical Officer, for his kind cooperation.

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ABSTRACTS OF ROENTGEN AND RADIUM LITERATURE

ROENTGEN DIAGNOSIS

HEAD

HEMPHILL, JAMES E. A simple technic for cerebral arteriography. *Radiology*, Oct., 1942, *39*, 432–436.

A simple technique for cerebral arteriography is described and illustrated with roentgenograms. The internal carotid on the side of the suspected lesion is exposed through a small incision under intravenous anesthesia. A silk thread is passed under the artery to stabilize it during the injection of the contrast medium. On the roentgen table the side opposite the exposed artery is placed toward the film which is in position in the Bucky carriage. Two other films are available near by. A needle attached to a syringe containing 20 cc. thorotrast is inserted into the exposed artery. Exposure is delayed for two or three seconds after blood enters the syringe and then the first film is exposed. This shows the outline of the internal carotid to the level of the anterior, middle and posterior cerebral vessels. The two subsequent films are exposed as quickly as possible. The three exposures can be completed in eleven to twelve seconds. The second film shows most of the smaller branches of the anterior, middle and posterior cerebral arteries. The third shows the smallest cerebral arteries, the venous pools and usually the larger arteries still filled with the contrast medium. The wound is closed while the films are being developed. The hemorrhage from the puncture wound in the artery can be controlled by sponge pressure.—*Audrey G. Morgan.*

DANDY, WALTER E. Intracranial arterial aneurysms in the carotid canal; diagnosis and treatment. *Arch Surg.*, Sept., 1942, *45*, 335–350.

A case of large aneurysm of the carotid artery beginning in the carotid canal and extending into the skull is described. The patient was a woman of fifty-two whose symptoms began with diplopia followed by severe pain in the right eye, piercing headache in the right frontal region, nausea and vomiting, and ptosis. The most important signs of an aneurysm in this region are palsy or paralysis of the third nerve and severe pain in the affected eye or frontal region. These signs may also occur, however, in intracranial aneurysms of the internal carotid or even of the posterior communicating artery. But when there is also subjective or objective involvement, or both, of the first and second branches of the trigeminal nerve, as in this case, the diagnosis of aneurysm in the carotid canal is almost certain. Angiography has been used for the diagnosis of these tumors but the author advises against it, as it is not free of danger.

In this case operation was performed successfully by establishing adequate collateral circulation in the circle of Willis by preliminary partial ligation of the carotid in the neck and then trapping the aneurysm by placing a silver clip on the internal carotid inside the skull and ligating the artery with silk in the neck. The author had previously used this method in smaller aneurysms but feared it would not be effective in one as large as this. Pulsation of the aneurysm stopped within five minutes. Two months later the patient was free of all symptoms except that the exophthalmos had not decreased.

A table is given showing the outstanding features of 35 similar cases reported in the literature. Among the 7 patients operated on 1 died, a mortality of 14 per cent. The other 6 patients recovered, the longest interval since operation being three and a half years.—*Audrey G. Morgan.*

DYKE, CORNELIUS G. Acquired subtentorial pressure diverticulum of a cerebral lateral ventricle. *Radiology*, August, 1942, *39*, 167–174.

In the condition described in the title a part of the medial portion of the atrium of a lateral ventricle is pushed medially and caudally through the incisure in the tentorium and forms a sac filled with fluid beneath the tentorium. If care is not used in diagnosing this condition it may be mistaken for an enlarged fourth ventri-

cle displaced rostrally, which would lead to mistaken diagnosis and treatment.

Three cases of this form of diverticulum are described, in 2 of which the increased intracranial pressure that led to the condition was caused by brain tumors. The first was a case of astrocytoma of the mid brain with stenosis of the aqueduct in a man aged twenty-nine, the second a case of parasagittal meningioma in a woman aged forty-six and the third a case of an obstructing scar in the aqueduct of Sylvius in a girl aged sixteen. In 2 cases the diagnosis was made from pneumencephalograms, which are reproduced.

The nature of the wall of these diverticula is not known except that it probably consists of a thin layer of brain tissue and leptomeninges and may be lined with ependyma. A detailed discussion is given of the site of the brain defect in these cases, the way in which it is brought about and the importance of its recognition. —*Audrey G. Morgan.*

ANDERSON, FRANK M., and ADELSTEIN, LEO J. Ganglion cell tumor (ganglioglioma) in the third ventricle. *Arch. Surg.*, July, 1942, *45*, 129–139.

Since interest in the subject has increased in recent years it is known that ganglion cell tumors of the brain are not so rare as they were formerly thought to be.

The authors describe a case of ganglioma of the third ventricle in a boy nine years of age seen at the Los Angeles County Hospital. He had shown gradual impairment of vision for three years with progressive development of headache, somnolence and vomiting. Mind and personality were not affected. There were no disturbances of gait and no tendency to stagger. Ventriculography showed bilateral symmetrical internal hydrocephalus with no air at all in the third ventricle. Operation showed a large ganglioma of the third ventricle which was completely removed and the patient made an uneventful recovery

There is no pathognomonic clinical picture in these tumors and ventriculography should be performed in any case in which there are good reasons to suspect such a tumor. The technique of the surgical operation is described. A frontal approach through the dilated lateral ventricle is indicated unless the tumor is in the posterior part of the ventricle when it must be approached from above through the corpus callosum.—*Audrey G. Morgan.*

NECK AND CHEST

DICKES, R., KNUDSEN, A. F., and FRANCO, S. C. Multiple polyps of the esophagus. *Arch. Int. Med.*, July, 1942, *70*, 121–131.

A case of gastrointestinal hemorrhage from polyps of the esophagus is described. This is an extremely rare condition and only one other case is described in the literature.

The patient was a girl of ten when first admitted to the hospital suffering from hemorrhage. The diagnosis at that time was thought to be Banti's disease with hemorrhage from esophageal varices. Splenectomy controlled the hemorrhage temporarily but, in spite of the fact that the liver did not show evidence of permanent damage and portal pressure was not increased, the hemorrhages recurred. The taking of a biopsy specimen was followed by very severe hemorrhage. The specimen showed many blood spaces in the thickened squamous epithelium.

Finally five years after the beginning of the disease and after repeated esophagoscopic examinations a diagnosis of polyps of the esophagus was made. The polyps were treated by fulguration and the patient has been free of hemorrhage since that time.

There is some difficulty in the differentiation of esophageal polyps and esophageal varices after giving barium sulphate. Both appear as translucent areas in the lumen of the esophagus. Varices, however, show momentary variations probably resulting from peristalsis, while these polyps showed no such variations. This may prove a useful point in differential diagnosis. —*Audrey G. Morgan.*

TAYLOR, HENRY K., and SHULMAN, ISIDOR. Cardio-angiography. *Radiology*, Sept., 1942, *39*, 323–333.

Cardio-angiography is a method of examining the heart by injecting a contrast solution and making roentgenograms after periods calculated from the results of circulation tests. In 13 of 15 cases the circulation time of the right heart was approximately five seconds. That of the left heart is less constant.

This article is based on the study of a group of 15 patients, 4 of whom had rheumatic heart disease, 1 coronary thrombosis, 2 hypertension, 2 arteriosclerotic heart disease, 3 syphilitic heart disease and 3 bronchial asthma. A 70 per cent diodrast solution was used as contrast medium. Roentgenograms of normal hearts and

these various pathological conditions show a number of differences in the heart outline from that described in the classical texts.

This method should not be used in all heart cases, but only in those with unusual diagnostic problems. It is valuable in the diagnosis of pericardial effusions, arterial or ventricular aneurysms, constrictive occlusion and thrombosis of the superior vena cava and in differentiating cardiovascular from other mediastinal lesions. There were no serious reactions in any of the author's cases. The method is not recommended in patients who are acutely ill or who have severe decompensation or respiratory difficulty.—*Audrey G. Morgan.*

Peery, Thomas M. Incomplete rupture of the aorta; a heretofore unrecognized stage of dissecting aneurysm and a cause of cardiac pain and cardiac murmurs. *Arch. Int. Med.*, Nov., 1942, *70*, 689–713.

Incomplete rupture of the aorta is really a preliminary stage of dissecting aneurysm. Approximately the inner two-thirds of the intima are ruptured, the rupture usually occurring in the ascending part of the aortic arch at or near one of the commissures. The essential predisposing cause of incomplete rupture is severe hypertension continued for a long time. The precipitating cause may be strain or exertion.

Illustrative cases found on autopsy are described, with anatomical drawings and electrocardiograms. If dissection does not occur and the tear is well above the aortic commissures healing usually takes place. But if the rupture is at or just above a commissure and transverse to it, it usually causes aortic insufficiency and possibly death from congestive heart failure.

There are some points that may possibly be helpful in making an antemortem diagnosis of this condition. If a patient has had hypertension for a long time and suddenly has an attack of suffocation and dyspnea, which may or may not be accompanied by substernal pain, and there are systolic or diastolic murmurs or both, which had not been present before, it is suggestive of incomplete rupture, especially if the murmurs are harsh or rasping.

If the patient is seen for the first time during an acute attack and there are no murmurs a mistaken diagnosis of coronary thombosis may be made. The sudden onset of symptoms may suggest rupture of an aortic valve cusp.

In dissecting aneurysm the pain is usually more severe and the severity and location of the pain change as the dissection progresses. If the patient is first seen during heart failure caused by aortic insufficiency diagnosis is very difficult. If the symptoms begin suddenly, pressure is very high and there are no signs of syphilitic or rheumatic infection it may be possible to diagnose incomplete aortic rupture as the cause of the insufficiency.

Roentgen examination usually shows widening of the aortic arch but no signs pathognomonic of incomplete rupture.—*Audrey G. Morgan.*

Tager, Stephen N. Use of over-penetrated film technic in diagnosis of cavities. *Radiology*, Oct., 1942, *39*, 389–394.

The author emphasizes the value of the over-penetrated film technique in the demonstration of cavities in tuberculosis. He has used a very simple method of over-penetrated film technique that is available to any roentgenologist while laminagraphy is not available because of its prohibitive cost. In cases that show only a homogeneous veiling with the routine technique the over-penetrated technique may definitely reveal the presence of cavities. A series of routine and over-penetrated films is given illustrating this point.

The factors for the routine technique were 75–85 kv., 100 ma., time 1/10 second, distance 72 inches, rectification mechanical. For the over-penetrated film 50–60 kv., 100 ma., time ½ to 1 second, distance 40 inches, Bucky-grid ratio 5–1, rectification mechanical. It is to be noted that the Bucky grid is used. The term Bucky film is synonymous with over-penetrated film.—*Audrey G. Morgan.*

BLOOD AND LYMPH SYSTEM

Erf, L. A. Clinical studies with the aid of radiophosphorus. II. The retention of radiophosphorus by tissues of patients dead of leukemia. *Am. J. M. Sc.*, April, 1942, *203*, 529–535.

A study was made of the concentration of radiophosphorus in 32 patients dead of leukemia, 1 of neuroblastoma and 1 of chloroma. The technique of making the determinations is described and a table given showing the amount and dates of the doses of P^{32} given and amounts retained in the various tissues and organs in

microcuries per gram of wet weight of tissue assayed.

Because of the innumerable differences in age, weight, basal metabolic rate and diet of the patients, differences in the types of leukemia and in the amounts and routes of administration of the radiophosphorus given, it is hard to draw definite conclusions, but in general this work confirms that previously done on mice and shows that the greatest concentration of radiophosphorus is in the tissues which are most frequently infiltrated with leukemic cells.—*Audrey G. Morgan.*

Pascucci, Lucien M. Chronic leukemia; statistical study of symptoms, duration of life, and prognosis. *Radiology*, July, 1942, *39*, 75–80.

This study is based on 64 patients with myeloid and 64 with lymphatic leukemia treated by roentgen irradiation at the Presbyterian Hospital in New York between 1919 and 1939. Cases in which the duration of life was less than four months from the beginning of symptoms were considered acute and are not included in the study. All patients with chronic leukemia were given roentgen treatment. Irradiation of the spleen was used in all except two cases in which general irradiation of the body was given. At first medium voltage was used, later high voltage treatment: 200 kv., 8 or 25 ma., 50 cm. distance and 1 mm. copper and 1 mm. aluminum filter. The skin over the spleen was given 25 to 50 r once or twice a week for several weeks, after which a dose of 75 r was given once a week. If there were enlarged peripheral lymph nodes they were given 50 r, which after one or two weeks was increased to 100 r.

Weakness and loss of weight occurred about one and one-half times as frequently in patients with myeloid leukemia as in those with lymphatic leukemia. There was a high incidence of splenomegaly in the myeloid form and almost as high in the lymphatic. The incidence of fever and hemorrhage was about the same in the two types. Retinitis was four times as frequent in the myeloid as in the lymphatic type.

The duration of life in chronic leukemia varies from two to four years according to reports in the literature but the authors found an average of 2.5 years in myelogenous and 2.8 years in lymphatic leukemia. It has not been definitely proved that roentgen treatment lengthens life but it certainly brings about remissions during which the patient may lead a normal and efficient life. And occasionally it seems life is prolonged.

An early fatal outcome is likely if the platelet and red cell counts are very low when the patient comes for treatment. Graphs are given showing the relation of survival to red blood count, platelet count and primitive cell count in the two types of leukemia. The more chronic the disease, the greater the life expectancy. The question of whether acute and chronic leukemia are really the same disease is discussed.—*Audrey G. Morgan.*

Vieta, J. O., Friedell, H. L., and Craver, L. F. Survey of Hodgkin's disease and lymphosarcoma in bone. *Radiology*, July, 1942, *39*, 1–15.

The authors present data and conclusions based on a study of 38 cases of Hodgkin's disease and 15 cases of lymphosarcoma, in all of which there were roentgen signs of changes in the bones. A postmortem study was made of 47 cases of Hodgkin's disease and 54 cases of lymphosarcoma. There were bone changes in 49 per cent of the former and 29 per cent of the latter. A roentgen review of 257 cases of Hodgkin's disease showed 14.8 per cent with bone involvement and a similar study of 213 cases of lymphosarcoma showed bone lesions in 7 per cent. The incidence of bone changes in both these series is probably lower than the actual facts due to inadequate examinations. The bones most frequently affected were those of the pelvis, the vertebrae, ribs and femora; in most cases more than one bone was involved.

Involvement of bone in both these diseases takes place either by direct extension from affected lymph nodes or through the blood circulation. The most frequent clinical signs of bone involvement were localized pain and tenderness but sometimes there were also tumors around the edges of the bones and neurological changes caused by extension of vertebral lesions into the spinal canal. In both diseases a high serum phosphatase content seems to indicate involvement of bone. This change was found most frequently in cases in which the bone changes were osteoplastic. In Hodgkin's disease 57 per cent of the cases showed a combination of osteoplastic and osteolytic changes, while in lymphosarcoma the changes were entirely osteolytic in 85 per cent of the cases. The character of the bone lesions is

different in the two diseases but not sufficiently so as to make a sharp differentiation between the two types possible.

The bone changes may occur at any time in the course of the disease. They are more apt to be terminal in lymphosarcoma as in this disease they occurred in 63 per cent of the cases in the last third of the course of the disease while in Hodgkin's disease they occurred in two-thirds of the cases earlier than that.

Irradiation with moderate doses often brings about marked improvement in the symptoms of Hodgkin's disease and in some cases restores normal bone structure. There was sometimes clinical improvement in lymphosarcoma also but death occurred so soon after the beginning of the bone changes that it was impossible to study the changes in the bones brought about by the treatment.—*Audrey G. Morgan.*

GENERAL

CAHILL, GEORGE F., MELICOW, MEYER M., and DAREY, HUGH H. Adrenal cortical tumors; types of nonhormonal and hormonal tumors. *Surg., Gynec. & Obst.*, Feb., 1942, *74*, 281–305.

This is a report of the adrenal cortical tumors which have been seen at the Squier Clinic (Department of Urology and Biochemistry, College of Physicians and Surgeons, Columbia University).

Tumors of the adrenal may be classified as follows:

(1) Tumors from the connective tissue, blood vessels, lymphatics, or nerve structures such as may occur in any gland and are not peculiar to the adrenal. These occur rarely.

(2) Tumors derived from the medulla which are peculiar to that tissue and are derived from the ectodermal sympathetic nerve system. They may be of the basic nerve cell, or sympathoblastoma; from the sympathetic ganglion or ganglioneuroma; from the pheochromocytes or paragangliomas. The first two are seen more frequently in children and are more common than the paragangliomas which occur more often in adults.

(3) Tumors originating from the adrenal cortex are mesodermal glandular tumors and are, cytologically, hyperplasias, adenomas, or carcinomas.

Accessory adrenals occur and are called Marchand's bodies. These are made up of cortical tissue and they are rare.

The Adrenal Cortex and Tumors of the Adrenal Cortex. The adrenal cortex has been definitely established as the elaborator of hormonal substances some of which are necessary for life maintenance. Chemically, these hormonal substances have been shown to be sterol lipoids. The adrenal cortical tumor occurs relatively infrequently. The clinical manifestations of these tumors may be as follows:

(1) No recognizable hormonal changes
(2) Changes due to excess androgens
  a. In female child toward adult masculinity
  b. In female adult toward masculinity
  c. In male child toward adult masculinity
(3) Changes due to excess estrogens
  a. In adult male toward femininity
(4) Changes due to excess androgens and other steroids
  a. Cushing's syndrome with associated sexual changes (mostly in females)
(5) Changes due to excess other stercids related to metabolism
  a. Cushing's syndrome without sexual change (in male and female)

Diagnosis and Treatment of Adrenal Cortical Tumors.

(1) The status of the adrenals may be adequately shown by air insufflation roentgenograms.

(2) Removal of adrenal tumors by the transperitoneal route is surgically best.

(3) Acute adrenal deficiency occurs only in those patients with symptoms described as Cushing's syndrome.

(4) The therapy of acute adrenal deficiency is similar to that which is used in the acute deficiency crisis in Addison's disease.

(5) Histologically, the tumors that produce the hormonal syndromes have cytoplastic lipoid vacuoles in amounts comparable to the symptoms.—*Mary Frances Vastine.*

ROENTGEN AND RADIUM THERAPY

BUTLER, FRANK E., and WOOLLEY, IVAN M. The present status of roentgen therapy in chronic paranasal sinusitis. *Radiology*, July, 1942, *39*, 69–74.

The authors first reported a series of 100 cases of chronic sinusitis begun in 1929; their results were greeted with a great deal of skepticism by

rhinologists and the method severely criticized. Now, however, they report 450 cases treated up to 1934 with 36 per cent of the patients free of symptoms, and 55 per cent improved. Compilation of the statistics of six observers shows an average of 33 per cent symptom-free, 41 per cent improved and 26 per cent unimproved. They have never yet seen any harmful results from the treatment and it does not cause fibrosis which would interfere with later operation as was predicted.

Illustrations are given of the masks used to protect the part of the face that is not to be irradiated. The authors prefer the single massive dose technique though equally good results have been obtained by other workers with the fractional dose method. They believe more depends on the skill of the operator than on the technique. They find a 200 kv. technique superior to a 120 kv. one because the greater filtration gives a wider margin of freedom from skin reactions; it also makes it possible to give a greater amount of radiation through the sphenoid sinuses. They now use 200 kv., 20 ma., through the equivalent of 0.75 mm. copper and 3 mm. aluminum filter at a distance of 50 cm., half-value layer 0.02 mm. copper; a dose of 300 r measured in air is given on each side and the patient told to return in six to eight weeks if he has further symptoms.

Roentgen treatment is not successful in allergic sinusitis. The best results are seen in chronic infections with a markedly thickened membrane and a small central pneumatized cavity. —*Audrey G. Morgan.*

FRIEDMAN, MILTON, and FINKLER, R. S. The treatment of sterility with "small dose" x-ray therapy. *Am. J. Obst. & Gynec.*, May, 1942, *43*, 852–857.

A case report is given which strongly suggests that small dose roentgen therapy can favorably affect sterility due to ovarian deficiency. In 1935, the patient's sterility was accompanied by irregular menses, possible estrogen deficiency (as evidenced by bio-assays), and faulty luteinization (as evidenced by endometrial biopsies). The first course of small dose roentgen therapy was followed by normal menses and two pregnancies within nine months. The first pregnancy was accidentally interrupted by a pre-menstrual biopsy which removed a twelve to fourteen day old embryo. The monthly cyclical bleeding was not thereby disturbed. The second pregnancy resulted in the delivery of a normal child at full term. Four years later, the clinical picture of ovarian deficiency returned in a more severe form, as evicenced by amenorrhea and absent luteinization. A second course of small dose roentgen therapy was followed by restoration of normal menses and a third pregnancy with delivery of a normal child.

Both courses of roentgen therapy were the same. Four treatments were given over a seven to ten day interval. A total dose of 80 roentgens (measured with back scattering) was given to the pituitary gland and 80 roentgens was given to each ovary.—*Mary Frances Pastine.*

SEWELL, ROBERT L., DOWDY, ANDREW H., and VINCENT, JAMES G. Chemotherapy and roentgen radiation in Clostridium welchii infections. *Surg., Gynec. & Obst.*, Feb., 1942, *74*, 361–367.

Fifteen patients with "gas gangrene" have been seen in the Strong Memorial Hospital, Rochester, N. Y., since 1937. The authors' work is based on the experience with these 15 cases together with the study of 255 dogs in which they produced *Clostridium welchii* infection experimentally.

Clinical clostridium infections range in severity from relatively benign infections to more fulminating and malignant types. In this work the criteria for calling cases "gas gangrene" were: (1) positive cultures of *Clostridium welchii;* (2) spreading infection; (3) gas in the tissues, and (4) discoloration and edema of the involved structures.

The summary of the findings is outlined as follows:

(1) Many of the virulent infections caused by *Clostridium welchii* are more of the nature of a cellulitis than a gas gangrene.

(2) Radical surgery is recommended only in cases of severe vascular damage.

(3) Sulfanilamide or roentgen irradiation, when used alone, exerts a beneficial effect on the course of the disease, and to about the same degree. Sulfathiazole is probably somewhat more efficacious. Sulfadiazine is superior to any other drug or method of treatment (experimental proof only).

(4) The combined use of both sulfadiazine and roentgen irradiation has given poorer results experimentally than has the use of either separately.

(5) *Clostridium welchii* antiserum is of some

efficacy only when additional methods are used to combat the local lesion.

(6) Conservative surgery coupled either with a sulfonamide or with roentgen irradiation is the treatment of choice. At present, conservative surgery plus sulfadiazine is recommended. —*Mary Frances Vastine.*

Cosbie, W. G. The complications of irradiation treatment of carcinoma of the cervix. *Am. J. Obst. & Gynec.*, Dec., 1941, *42*, 1003–1008.

The hope of increasing the salvage rate of patients suffering from carcinoma of the cervix depends on carrying radiation treatment to a point sometimes beyond the tolerance of the adjacent tissues. This is true because 65 to 70 per cent of patients suffering from carcinoma of the cervix do not come for treatment until the disease has progressed to Stage III or IV. Two types of reaction are met with: (1) the acute or inflammatory, and (2) the late or fibrotic which may pass on to a state of necrosis.

This study includes 320 patients treated for carcinoma of the cervix during the years 1936 to 1940 inclusive. The complications which can occur may be outlined as follows:

(1) Irradiation sickness (with cystitis and proctitis) is a fairly frequent development during or immediately following treatment.

(2) Cystitis is much less frequent when a self-retaining catheter keeps the bladder empty during radium treatment. Distortion of the base of the bladder with displacement of the ureteral openings is commonly observed.

(3) Carcinomatous infiltration of the bladder was found a number of times on cystoscopic examination when its presence had not been suspected. Seventeen of the 14 autopsies held on patients in this series showed ureteral obstruction, hydroureter and hydronephrosis or pyonephrosis. In every case, the obstruction was due to malignant infiltration of the cellular tissue.

(4) Fever and infection during the course of treatment may cause much concern. Out of 47 patients in the study with positive hemolytic streptococcus cultures, 25 developed some complication subsequently such as excessive pyrexia, vaginitis, pyometria, cellulitis, thrombophlebitis, pelvic abscess, peritonitis or septicemia.

(5) Proctitis is encountered frequently but it is a condition which ordinarily disappears within a few weeks. The typical late irradiation lesion is rectal ulceration. Two types have been described: (*a*) primary chronic ulceration of the rectum, and (*b*) an extrinsic type which is characterized by massive perirectal cellulitis and stenosis of the rectum as a result of which ulceration develops. The warning is given that symptoms simulating intestinal obstruction several months or years after irradiation may well be due to stricture rather than metastases.

(6) Spontaneous fracture of the neck of the femur following roentgen treatment of carcinoma of the cervix can occur but none of the patients in the series under discussion experienced this complication.—*Mary Frances Vastine.*

Desjardins, Arthur U. Problems in roentgen therapy for Hodgkin's disease and lymphosarcoma. *Radiology*, July, 1942, *39*, 16–26.

Often the maximum results are not obtained in treating Hodgkin's disease or lymphosarcoma with roentgen rays. This may be because the rays have not been applied to the fields where the cause of the trouble is really located; it may be due to improper time distribution of the treatment, wrong quantity or quality of rays, pathological factors or clinical factors that depend on the stage of the disease. There is a general tendency to direct treatment to the cervical, axillary and inguinal lymph nodes but there may be involvement of the mediastinal or abdominal or retroperitoneal nodes and the fields must be selected accordingly. Irradiation of the abdomen affects the leukocytes more than that of any other part of the body so the leukocyte count must be watched very closely during abdominal irradiation.

From the author's experience with more than 2,000 cases of lymphoblastoma he believes that irradiation at 140 kv. is more effective than that at 200 kv., provided the treatment is applied to the correct anatomical fields and excessive doses are not given. This is true even of mediastinal and retroperitoneal lymphoblastoma.

The doses required are not as large as those for epithelioma. Therefore protracted fractional irradiation is not necessary. The best long-terms results are obtained by giving each field in one or two days as large a dose as can be tolerated without injuring the skin, usually 550 to 600 roentgens, surface dose measured in air. The fields are treated successively one at a time until all have been given the required dose. This depends on the extent of the involvement and the tolerance of the patient.

Failure may be due to giving too small doses at each treatment or leaving too long intervals between treatments. Or it may be due to pathologic factors such as beginning treatment in the terminal stage of the disease. Special problems may be created by lymphoblastomatous infiltration of the skin, subcutaneous tissues, abdominal organs, rectum, orbit, meninges, ureter or kidney or suprarenal glands or erosion of a bronchus, which may result in fatal hemorrhage if not treated in time. The symptoms of these various types of infiltration are discussed and the importance of early detection and treatment stressed.—*Audrey G. Morgan.*

RANDALL, LAWRENCE M., LOVELADY, SIM B. and SLUDER, FLETCHER S. Radium in the treatment of uterine bleeding caused by benign lesions. *Am. J. Obst. & Gynec.*, March, 1942, *43*, 377–387.

Complete follow-up records were studied on 196 women who experienced atypical uterine bleeding (menorrhagia and/or metrorrhagia) caused by benign conditions, five or more years after treatment by radium or roentgen rays. One hundred seventy-five patients were treated with radium and 21 with roentgen rays. The bleeding was satisfactorily controlled by the initial treatment in 134 (68.4 per cent). In 13 cases subsequent irradiation was necessary before bleeding was controlled. Thus good results were obtained in 147, or 75 per cent. Of the 125 patients who were forty years of age or younger and treated with radium, 21 (16.8 per cent) established a fairly normal menstrual cycle after radium therapy. The percentage of good results from all types of radium treatment increased with the age of the patients. As the age of the patient increased, the dose of radium administered tended to increase. The authors believe that for patients more than forty years of age without fibroids or when fibroids are present but the uterus is not enlarged to more than its size after three months of pregnancy, preliminary dilatation and curettage for diagnosis followed by intrauterine application of a menopausal dose of radium or the administration of roentgen therapy are excellent forms of treatment for atypical uterine bleeding. For the group of patients who are from thirty to forty years of age, the choice of treatment is made with more difficulty. To one who is surgically minded, failure of about 25 per cent from irradiation in the entire series would indicate that hysterectomy accompanied by a 98 to 99 per cent possibility of cure is preferable.

The dosages of radium and roentgen radiation given these 196 patients are indicated in tables included in the article. Of the patients forty years of age or less without myomas (79), some received less than 500 mg-hr., others 500 to 1,000 mg-hr. and another group 1,000 to 2,000 mg-hr. or more. The doses of radium in the 35 patients forty years of age or less with myomas varied from 500 mg-hr. to 1,000–1,200 mg-hr. or more. The patients who were more than forty years of age with or without myomas (47) received 1,000–1,200 or more mg-hr. of radium. The total roentgen-ray dosage administered to 21 patients varied from 700 to 1,760 r. —*Mary Frances Vastine.*

STEVENS, ROLLIN H. Radium poisoning. *Radiology*, July, 1942, *39*, 39–47.

A case of radium poisoning is reported in a man of thirty-six, a commercial photographer who had always been well except for night sweats, which he had had since youth. In the spring of 1924 he first noticed enlarged lymph nodes on both sides of the neck and in the axillae and groins. Histological study of a piece excised from one of these nodes showed Hodgkin's disease.

Roentgen treatment was begun on July 29, 1924, and the size of the nodes decreased. But on January 27, 1925, roentgen examination of the chest showed continued enlargement of the mediastinum and it was concluded that the injection of radium was justifiable in order to cure the Hodgkin's disease. On January 21, 1925, 20 micrograms of radium chloride was injected. Injections were continued up to November 24, 1930. During this period of five years and nine months 440 micrograms was given. On June 4, 1938, a dentist discovered an area of necrosis in the jaw bone. This necrosis soon involved the right side of the mandible and the jaw became fixed. Suffering was severe for months. Measurements showed that the bone was radioactive. The patient was put on a calcium-free diet, thyroid extract and ammonium chloride which stopped the pain and resulted in great improvement. Subsequently he again suffered great pain; he was put on this treatment again and recovered again. Now after eighteen years of treatment he is well, is continuing his work and does not show any signs of Hodgkin's disease. His blood picture has re-

mained normal throughout the years except that occasionally there has been 4 to 5 per cent eosinophils. There is no sign of malignancy. He is happy and contented and says he would have taken the treatment for the cure of the Hodgkin's disease even if he had known it would caused the radium poisoning. The interest of this case lies in the fact that a known quantity of radium was given over a period of eighteen years. The amount retained was 11.4 micrograms out of 449 micrograms, or 2.5 per cent, more than enough for destructive action. —*Audrey G. Morgan.*

MISCELLANEOUS

Voegtlin, Carl. Trends in cancer research. *Surg., Gynec. & Obst.*, Feb., 1942, *74*, 561–564.

At the end of the nineteenth century, Claude Barnard's plea for controlled experimentation was applied to the cancer problem. As a result a large number of new facts have been discovered:

(1) The first important conclusion reached from experimental work was the high specificity of malignant cells. Each one of the many types breed true in vivo and in vitro.

(2) In 1915, the production of skin cancer in rabbits by the long continued topical application of coal tar was announced. This important discovery shifted the trend of cancer research into the field of chemistry.

(3) Recently some progress has been made in the study of the dietary factor in the production of induced tumors.

(4) The discovery of the artificial chemical carcinogens was a milestone in cancer research as it has made possible the experimental production of tumors under controlled conditions. It is firmly established that very large doses of female sex hormones acting over a long time can induce malignant tumors of the breast and uterus in mice and rats.

(5) Recently, claims of the carcinogenic action of cell free extracts of tissues and of urine from cancer patients have been made. Final proof of these claims must wait on more thorough investigation.

(6) Experiments with normal and regenerating rat livers and rat hepatomas make it appear that some of the important enzymatic activities of the tumor show marked decreases. This attribute may perhaps account for the specific tumor biology.

(7) A very great reduction in catalase activity in the hepatic tumor has been found while a lesser decrease has been noted in that part of the liver which is apparently normal in the tumor-bearing animal.—*Mary Frances Vastine.*

Rhoads, C. P. Precancerous lesions. *Surg., Gynec. & Obst.*, Feb., 1942, *74*, 549–551.

The common factor to be discerned in those changes which are known to tend to become neoplastic is probably an atrophy. This atrophy is probably due to some mechanism which prevents the normal life and reproduction of the affected cells.

A table in which are presented recognized examples of cancer which is known to follow the appearance of an atrophic process is reproduced:

PRECANCEROUS CHANGES

1. Skin
   - A. Keratoses of the atrophic skin
   - B. Xeroderma pigmentosa (atrophy)
   - C. Radiation dermatitis (atrophy)
   - D. Burn scars and lupus (atrophy)
   - E. Arsenic dermatitis (atrophy)
2. Kraurosis vulvae (atrophy)
3. Plummer-Vinson disease (atrophy)
4. Thyroid cancer after hypothyroidism
5. Cancer in undescended atrophic testicle
6. Cancer of liver on cirrhosis
7. Leucemia on aplastic anemia

Kinosita proved that the feeding of dimethylaminoazobenzene (butter yellow) to rats taking a diet of rice would regularly produce hepatic cirrhosis followed by hepatic cancer. This experiment was verified and further analyzed in the author's laboratory. It is observed that cancer recurs and grows in the face of a milieu which is incompatible with the life and growth of normal cells. Thus it has been proved that a concentration of a toxic breakdown product of butter yellow which will kill normal rat liver cells has no effect upon the growth and multiplication of liver cancer cells.—*Mary Frances Vastine.*

Lieberman, Louis M., Hodes, Philip J., and Leopold, Simon S. Roentgen therapy of experimental lobar pneumonia in dogs. *Am. J. M. Sc.*, Jan., 1941, *201*, 92–100.

The authors have pursued the study of roentgen therapy on experimentally produced lobar pneumonia for some three years, beginning just prior to the advent of chemotherapy. They continued these studies because of the very encouraging evidence.

Forty-five dogs were infected; 26 of these were treated with roentgen radiation; 19 serving as controls. The development of pulmonary disease was confirmed by roentgenoscopy and roentgenography.

The quality of radiation applied varied, including that produced by 80 kv. (5 mm. Al); that produced with 135 kv. (0.25 mm. Cu plus 1 mm. Al), and that produced with 200 kv. (0.5 mm. Cu plus 1 mm. Al).

Twenty of the animals were handled as group 1; half of these were treated with roentgen radiation, the other half serving as controls. The dosages varied from 70 to 120 r, one, two or three treatments being given at daily intervals. All of this group died. Group 2 included only 4 animals, 1 serving as control, the other 3 being treated with 135 kv. therapy. One of these survived (receiving 150 r the first day; 124 r the second). There were 21 animals in the third group, 8 of these serving as controls. The others were treated with 200 kv. therapy receiving treatments on the first, second or third days, varying from 107 to 240 r. Five of the 13 animals survived; those receiving, for the most part, the greatest dosage and treatments over the three days.

Pathologically, there were found pneumonic infiltrations, including congestion, edema, hemorrhage and neutrophile infiltration. Atelectasis was found in about one-half of the cases. The irradiated animals showed about the same degree of congestion and hemorrhage as found among the controls. However, those receiving roentgen radiation showed less edema, less atelectasis, a decrease in neutrophiles and a relative increase in round cell infiltration.

The authors speak of Korb's work which indicated that even with dosages as high as 22,000 r, using high voltage, there was no effect upon cultures of tubercle or colon bacilli; whereas, with rays generated at 50 kv. these bacteria were killed with smaller dosages. Korb also found that bacterial radiosensitivity increased markedly with increases in temperature. They also refer to those studies which indicate that irradiation increases the alkalinity of tissues and that an alkaline medium is more ideal for neutrophiles. Mention is also made of changes in the molecular structure of toxins as well as of allied proteins and the elevation of the globulin fraction (and the concurrent increase in antibodies). Reference is also made to Desjardins' theory of leukocytic degeneration by roentgen irradiation and the resultant stimulation to phagocytosis locally.—*A. A. de Lorimier.*

BEARD, J. W., TAYLOR, A. R., SHARP, D. G. and BEARD, DOROTHY. The nature of a virus associated with carcinoma in rabbits. *Surg., Gynec. & Obst.*, Feb., 1942, *74*, 509–513.

The isolation and properties of a specific material from virus-induced cottontail rabbit papillomas are described in this article. This material behaves biologically as does the virus responsible for the disease. It is a nucleoprotein obtainable in solutions of high homogeneity. Its physical properties with respect to sedimentation, electrophoresis, diffusion, and viscosity are those of a molecular substance. With respect to the physical state this animal virus falls into the same category as that occupied by the macromolecular plant virus nucleoproteins.—*Mary Frances Vastine.*

ELLINGER, FRIEDRICH, and DAVISON, CHARLES. Changes in the central nervous system of goldfish irradiated in the depths of a water phantom. *Radiology*, July, 1942, *39*, 92–95.

Goldfish have been found suitable animals for the evaluation of the biological action of roentgen rays. In this article experiments are described in which goldfish were irradiated in the depth of a water phantom measuring 32×32×32 cm., which permits of maximum backscatter even at a depth of 10 cm.

Histologic examination of the brains of these goldfish showed marked changes, particularly in the medulla oblongata. The technique of the experiments and the histological findings are described. A comparison of the findings in one of the fish irradiated in the phantom and another irradiated at the surface showed that though the animals survived for about the same length of time there were more marked changes in the myelin sheaths of the fish irradiated in the depths of the phantom. There were greater changes also in the nerve cells of the medulla oblongata; there was almost complete destruction of the cell with a dose of 1,000 r in the depths while a dose of 1,250 r on the surface caused only slight decrease in the number of cells. In one fish an estimated body dose of about 340 r caused pigmentation and brain changes at a depth of 10 cm., though the minimal dose required to cause pigmentation on the surface is about 800 r in air.—*Audrey G. Morgan.*

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1. Gardner, W. J., and Frazier, C. H. Ventriculography without air injection. J. Am. M. Ass., 1929, 93, 193–194.

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THE AMERICAN JOURNAL OF ROENTGENOLOGY AND RADIUM THERAPY

VOL. 51 MARCH, 1944 No. 3

THE DEMONSTRATION OF PULMONARY LOBES AND INTERLOBAR FISSURES BY ROENTGENOGRAMS OF AN ARTIFICIAL THORAX

By E. M. MEDLAR, M.D., G. S. PESQUERA, M.D., W. H. ORDWAY, M.D., AND E. C. LASHER, R. T.

From the Hegeman Research Laboratory and the Metropolitan Life Insurance Company Sanatorium

MOUNT MCGREGOR, NEW YORK

IT IS the usual procedure to interpret the pathologic conditions observed in roentgenograms of the chest as being in the upper, middle or lower thirds of the pulmonary field, or in the hilar, central or lateral thirds. Such interpretations disregard the larger anatomical units, the lobes of the lung. Anyone who is familiar with the pathology of pulmonary tuberculosis in man should be acutely conscious of the importance of the large anatomical units of the lung, for in this disease the preponderance of dangerous cavitating lesions occurs in the cephalad or superior portions of the lobes, and tuberculous foci in the inferior or caudal portions show a very strong tendency to retrogress completely or to become small, firm, encapsulated lesions. Because of this peculiarity of pulmonary tuberculosis it is important to determine whether the initial lesions and the "spreads" of the disease are in the upper or the lower portions of the lobes of the lung.

The prominent position now held by thoracic surgery also calls for the location of pulmonary pathology in relation to the pulmonary lobes. If lobectomy is to be performed a preoperative location of the pathologic condition enables the surgeon to appraise the physiological after-effect dependent upon the volume of tissue to be removed. The removal of the relatively small right middle lobe, for instance, should not cause any undue over-compensation of the remaining lung tissue, whereas the removal of a large lower lobe might cause a serious physiological complication at a date far removed from the operative procedure. If thoracoplasty is under consideration and the pathologic condition can be demonstrated to be in the cephalic portion of the lower lobe, then it would seem to be a needless sacrifice of lung tissue to perform a total thoracoplasty when only a limited area need be compressed.

Before an interpretation of the relative position of pathologic conditions seen in roentgenograms of the chest is possible, it is necessary to demonstrate the limits of the lobes of the lungs and of the interlobar fissures in a three dimensional field in the normal chest. This is impossible because under normal conditions the interlobar fissures are not of sufficient density to cast a shadow that would demarcate the lobes.

It is well known from dissection of cadavers and from necropsy observations that interlobar fissures vary to a certain degree in both their extent and their position. For instance, the fissure between the upper and middle lobes of the right lung may vary from a small superficial depression to a complete separation of the lobes down to the bronchial tree at the hilum. Textbooks of anatomy offer little aid since the pulmonary lobes and their relation to bony landmarks are dealt with in a very sketchy manner. The absence of any satisfactory method of orientation at present makes it impossible to determine whether lesions in certain areas are present within a certain lobe or an interlobar fissure.

The only approach to a solution of this problem is upon an experimental basis. This was first attempted by Sampson, Heise and Brown[1] when they used wire gauze to locate the interlobar fissures inside the thorax of a skeleton and then took roentgenograms from various angles. Later Berry and Childs[2] attempted to demonstrate interlobar fissures in cadavers by

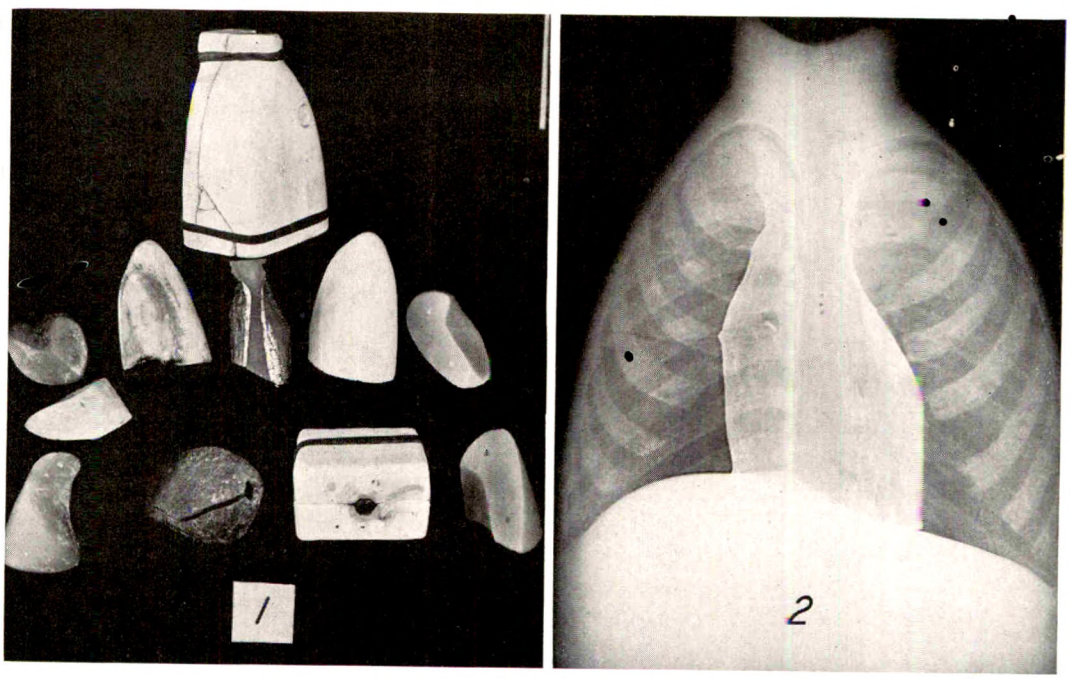


FIG. 1. This is an assemblage to illustrate the method of procedure and the casts used. At the top is the dummy set at an angle to show the anterior, posterior and lower divisions. It is assembled with the lung lobes and mediastinal contents in place and is ready for the taking of roentgenograms in any position desired. Just below the dummy is a paraffin cast of the mediastinal contents with the pleural surfaces covered with lead foil. To the left is the master plaster cast of the right lung as it appears from a medial view and to the right is the left lung as it appears from a lateral view. Below these structures there is a latex mold of a lobe of a lung and a mold assembled for the pouring of a cast of a lobe. Paraffin casts of the lung lobes are shown at the side—the three lobes of the right lung to the left and the upper and lower lobes of the left lung to the right. Throughout the study paraffin casts of the mediastinal contents and of lung lobes were used.

FIG. 2. A posteroanterior roentgenogram showing the mediastinal contents covered with lead foil. When considered *en masse* the trachea and main bronchi, the esophagus, the heart, the large arteries and veins, and the areolar and adipose tissue form a considerably greater bulk than is generally realized when viewing the usual chest roentgenogram. The apparently large size of the mediastinal contents as shown in this roentgenogram emphasizes that fact. If the appearance portrayed here is kept in mind when the other roentgenograms are studied a clearer conception of the various structures shown will be obtained and the blocking out by the mediastinal contents of certain portions of fissure and lobes can be appreciated.

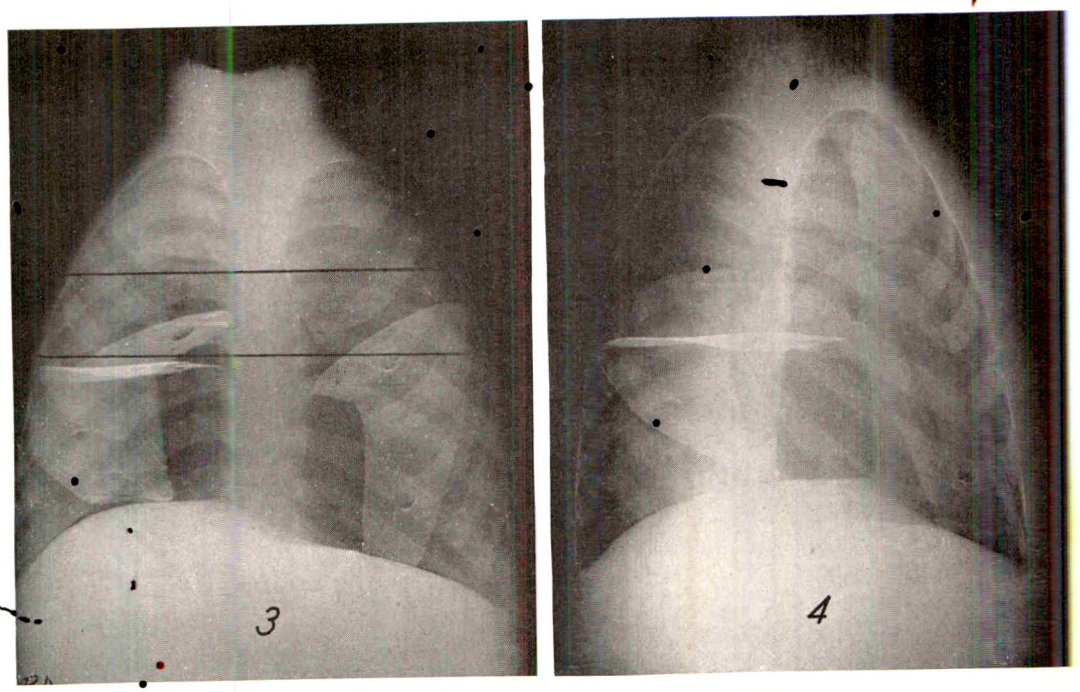


Fig. 3. A posteroanterior roentgenogram with lead foil placed in the three interlobar fissures. Note that in this pair of lungs the interlobar fissure on the left side is highest at the level of the fourth rib at the posterolateral part of the chest, while the analogous fissure on the right reaches its highest point at the level of the fifth rib at the costovertebral junction. The fissure between the upper and the middle lobe of the right lung has a slight upward concavity and extends almost horizontally at the level of the third rib ventrally. At full inspiration this level might readily be at a level between the third and the fourth ribs. The fissures follow the contour of the mediastinal contents and the major fissures extend down to the diaphragm. The two horizontal lines are drawn at the level of the fourth and the sixth costovertebral junctions and represent horizontal planes through the chest. The first rib shows only as a light line in this roentgenogram because of the angle at which the outline is pointed on the pleural surface. These points were chosen because they remain stationary, while the position of the ribs and the sternum changes somewhat during the process of breathing. Note the relationship of the interlobar fissures to the two horizontal planes. The practical significance of this arbitrary division of the pulmonary field is presented in the text and with the description of Figures 24 to 31.

Fig. 4. A right anterior oblique roentgenogram of the artificial chest shown in Figure 3.

placing lead foil between the pulmonary lobes *in situ*. This procedure proved to be none too satisfactory because only anteroposterior roentgenograms of the chest could be obtained. It was also found that the cooperation necessary to carry on such a study was difficult to obtain. Levitin and Brunn[3] carved model lobes of the lungs out of paraffin which offered little resistance to the passage of roentgen rays, bringing out the interlobar fissures by means of lead foil inserted between the lobes. These models were placed in position within the skeleton of a thorax and roentgenograms were obtained in various positions, such as posteroanterior, oblique and lateral. To illustrate the condition observed in the roentgenograms the authors resorted to diagrams as they did not consider the original roentgenograms satisfactory for reproduction. Such diagrams were none too satisfactory since the actual shape of the fissures and of the pulmonary lobes could not be appreciated due to the lack of an impression of depth in the diagrams. Peirce and Stocking[4] took roentgenograms of frozen sections of the thorax of cadavers which were cut in the right and left anterior oblique positions

and made careful comparisons between the roentgenograms and the corresponding section. These studies gave a fairly good idea of the lobes and fissures but they required reconstruction drawings to visualize the entire structures in these positions. In addition, the lungs in their specimens showed considerable disease and the thoraces were in a state of expiration so that the relationship to any bony landmark as seen in the usual roentgenogram would not be the same.

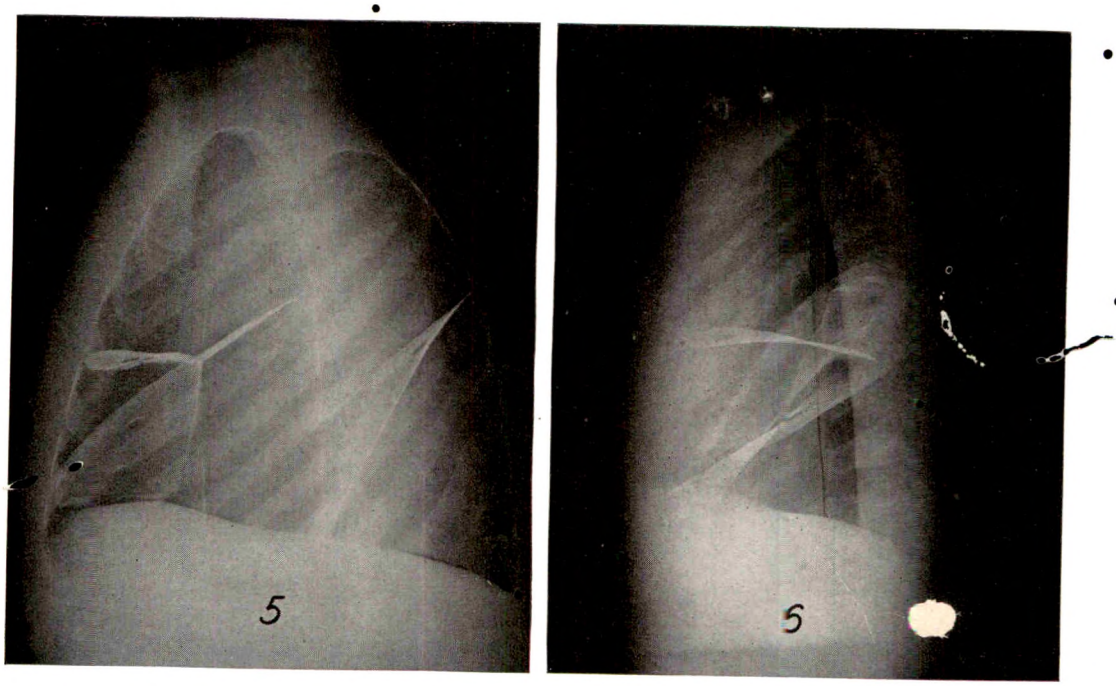


FIG. 5. A left anterior oblique roentgenogram of the same set-up as shown in Figure 3. Of particular interest is the considerable change in the configuration of the interlobar fissures when comparisons are made between Figures 3, 4 and 5.

FIG. 6. A left lateral roentgenogram of the model. This demonstrates clearly that the fissure between the right upper and middle lobes extends to the ventral but not to the dorsal costal surface. It is also to be noted that neither major fissure reaches to the ventral surface of the thoracic cavity while they both extend to the dorsal and to the diaphragm surfaces. The partial overlapping of the main fissures is plainly shown. This condition should be remembered when lateral roentgenograms of the human chest are interpreted.

It is practically impossible to obtain satisfactory molds or casts of entire lungs, or of lung lobes in the fresh state, because of the inherent nature of the tissue. For the same reason it is difficult to demonstrate the relationship of the interlobar fissures to the bony landmarks of the chest cavity, for normal lungs tend to contract to a certain extent when the thorax is opened, even in preserved cadavers. Any manipulation of the doughy tissue leads to artefacts which increase the difficulty of obtaining satisfactory roentgenograms in cadavers. The transference of entire lungs and of pulmonary lobes over to a solid substance that would permit of handling as one might choose would appear to be the procedure of choice in any study wherein anatomical relations are under consideration. It would also be desirable to use the same thorax from which the lungs are obtained but the acquisition of such a human thorax is next to impossible. No matter how such a study is carried out, artefacts cannot be avoided.

We conducted our study of this problem as follows: A pair of human lungs, free from

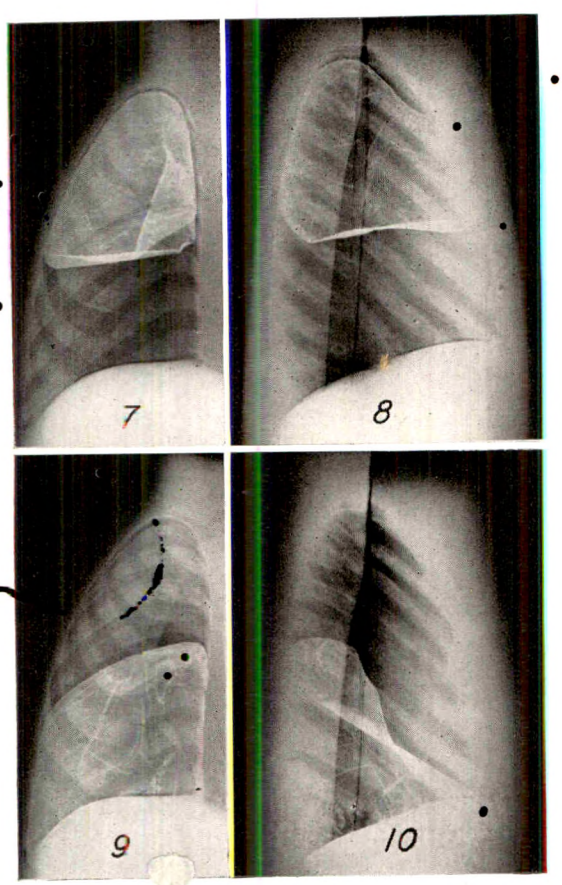


Fig. 7 and 8. A posteroanterior and a lateral roentgenogram of the right upper lobe covered with lead foil. The denser portions on the mediastinal and interlobar surfaces are due to several thicknesses of lead foil that the roentgen rays are obliged to penetrate and thus indicate the general contour of these surfaces. The notch in the upper portion of the lobe indicates the area occupied by the large vessels passing to the right arm and shoulder girdle. The diameter of the lobe is greatest in the dorsoventral axis and the interlobar surface has the shape of a check-mark with a short oblique dorsal and a long horizontal ventral portion.

Fig. 9 and 10. A posteroanterior and a lateral view of the right lower lobe. The mediastinal and diaphragmatic surfaces of this lobe are concave while the remainder of the outer surface is convex. The interlobar surface is twisted on itself and gives the appearance of a propeller blade when viewed laterally. The upper third is somewhat less perpendicular in its descent than the lower two-thirds. A considerable portion of the volume of this lobe lies below the highest level of the diaphragm. This area cannot be visualized in the posteroanterior view.

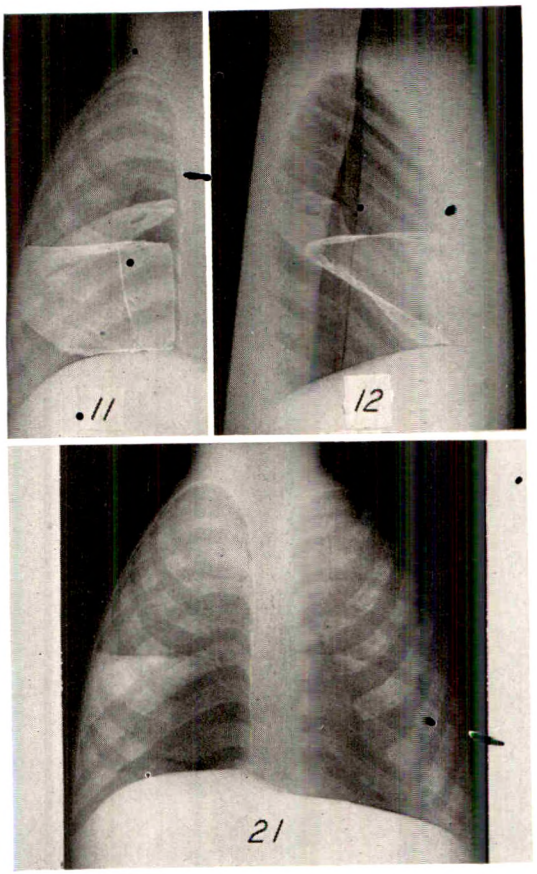


Fig. 11 and 12. A posteroanterior and a right lateral view with the right middle lobe and the main fissure covered with lead foil. This lobe has a wedge-like appearance when viewed from any angle. The mesial surface curves about the mediastinal contents in the ventral half of the chest. The volume of tissue ventral to the mediastinal contents as well as that portion resting upon the diaphragm is small. The dorsal tip of this lobe extends to about the mid-axillary line as is shown in the lateral view. The mediastinal surface is approximately of the same dimensions as the broadest lateral surface. The upper interlobar surface extends almost horizontally and is slightly convex. Lower interlobar surface is slightly concave and slightly twisted on itself to conform to corresponding portion of the lower lobe. Contact between the upper and lower lobes is dorsal and above the middle lobe.

Fig. 21. A posteroanterior view of an attempt to simulate a condition sometimes observed in roentgenograms of the chest. Note the wedge-shaped type of the "lesion" with the base in the lateral position. Such an appearance is commonly diagnosed as encapsulated interlobar effusion. Other views of this "lesion" are shown in Figures 22 and 23.

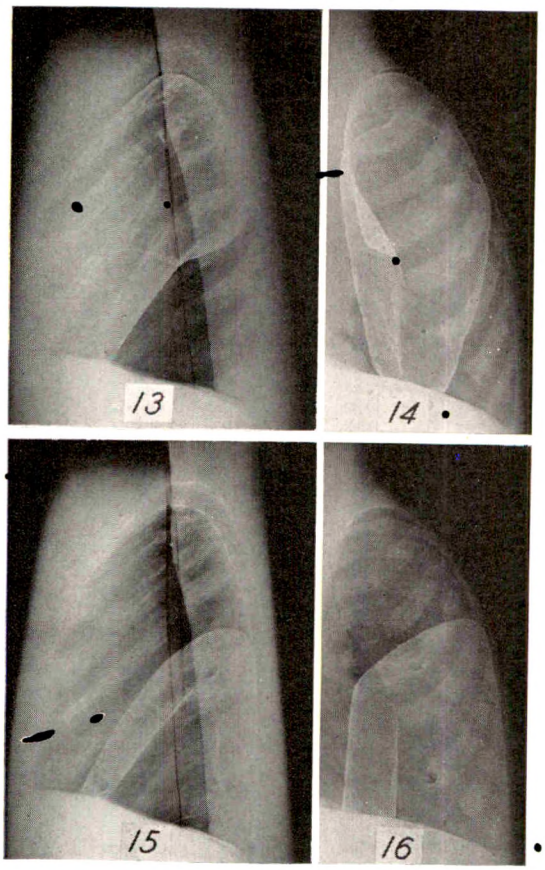


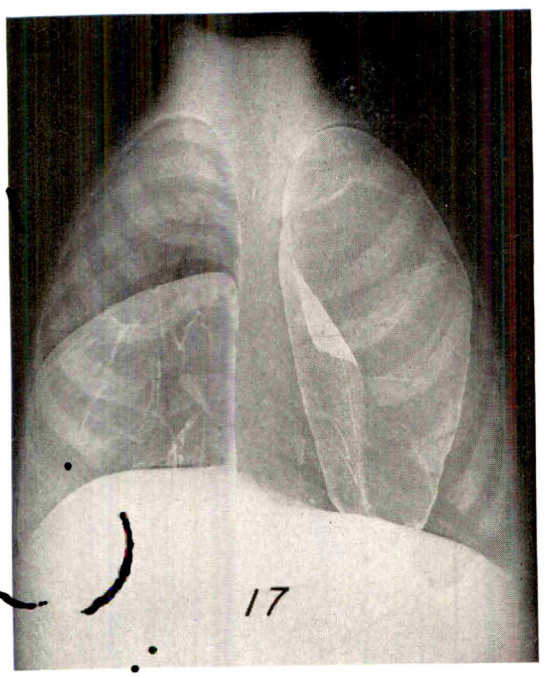
FIG. 13 and 14. These illustrate a left lateral and a posteroanterior roentgenogram of the left upper lobe. The greatest diameter of this lobe is vertical since it extends to the diaphragm although the volume of lung tissue is small at the diaphragm. The lobe extends around the mediastinal contents and is of small volume in the ventral flap. There is no cardiac notch, such as is usually described in textbooks of anatomy, in this particular specimen. The interlobar surface is slightly convex. About midway between the left anterior oblique and lateral positions this surface presents a linear appearance with a diagonal downward dorsoventral course of about 45 degrees.

FIG. 15 and 16. A left lateral and a posteroanterior view of the left lower lobe. The mediastinal and diaphragmatic surfaces are concave while the remainder of the outer surface is convex. A considerable volume of this lobe lies dorsal to the heart and is not seen in the usual posteroanterior roentgenogram of the chest. The same is true of smaller volume of tissue blocked out by the diaphragm. The interlobar surface is a reciprocal of the interlobar surface of the upper lobe.

macroscopic disease and with complete interlobar fissures on both sides, was obtained at necropsy.* This part of the program proved most difficult because of variations in the completeness of interlobar fissures and the presence of pleural or of interlobar adhesions. The lungs in the fresh state were moderately distended with 10 per cent formalin by the gravity method to approximate normal inspiration as nearly as one could judge with the lungs outside the thorax. In this condition they were immersed in 10 per cent formalin and thoroughly fixed for two weeks. The tissue was gradually dehydrated over a period of several months, starting with a 50 per cent solution of alcohol. The dehydrated lungs were placed in chloroform to displace the alcohol and later were transferred to a solution of chloroform saturated with paraffin. After several weeks in this solution the lungs were placed in melted paraffin in a paraffin oven, where many changes were necessary to get rid of the chloroform. The paraffin-impregnated lungs were then removed and quickly cooled to harden the paraffin. Because of the bulk of the tissue it took over a year to carry out the processes described. In the final process of hardening some irregular shrinkage of the lung surfaces, as well as of the entire lung, occurred since paraffin decreases in volume during the process of consolidation. These shrinkage defects were later remedied by molding modelling clay over the surface. During the whole procedure the interlobar fissures were not disturbed.

Plaster molds of each lung were made and master casts preserved. The lungs were then separated into lobes and molds and casts of each lobe obtained. Latex molds of the casts of the individual lobes were made and, by the use of these permanent molds, it is possible to obtain casts of any lobe as desired, either in plaster or paraffin. The master plaster casts of the whole lungs were placed in proper position and a plaster mold of the thorax was then cast with anterior, posterior and bottom portions. This was whittled down to crudely resemble the ex-

* We are indebted to Dr. J. Lebowich of the Saratoga County Laboratory for the pair of lungs we have used.



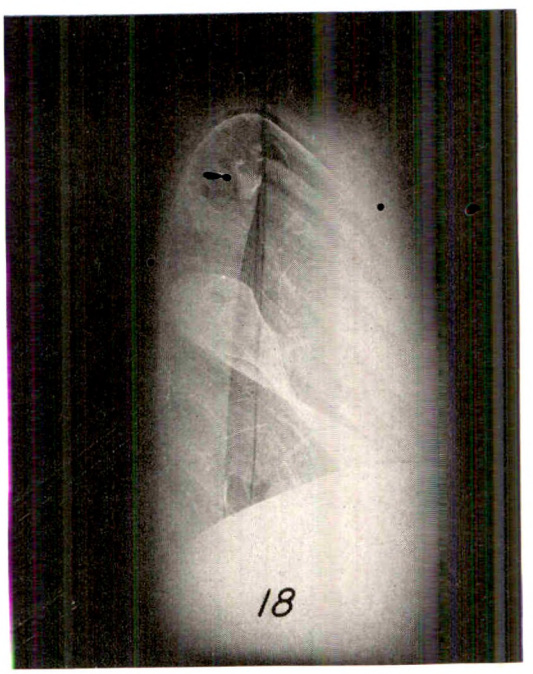
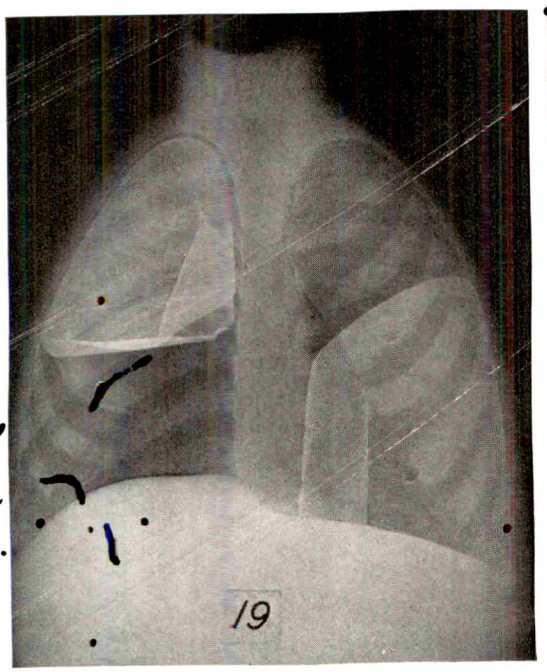


FIG. 17 and 18. A posteroanterior and a right lateral view with the right lower and left upper lobes covered with lead foil. Note the partial overlapping of the interlobar surfaces. The extent of this overlapping is shown more clearly in Figure 6.



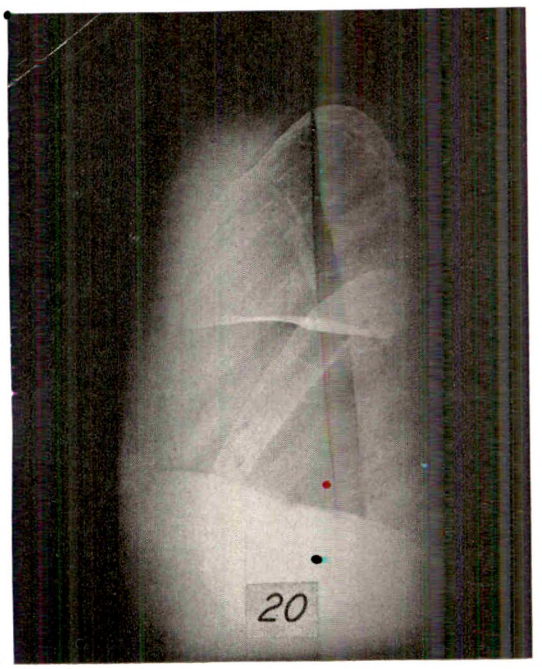
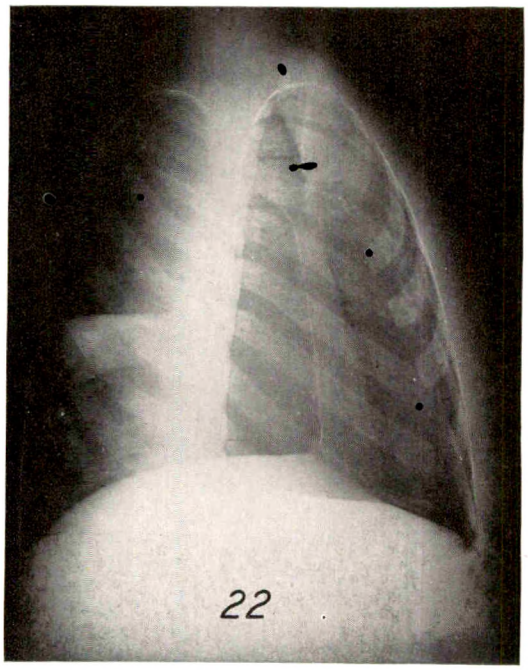


FIG. 19 and 20. A posteroanterior and a left lateral roentgenogram with the right upper and left lower lobes covered with lead foil. Of particular interest is the partial overlapping of the interlobar surfaces as shown in the lateral view.



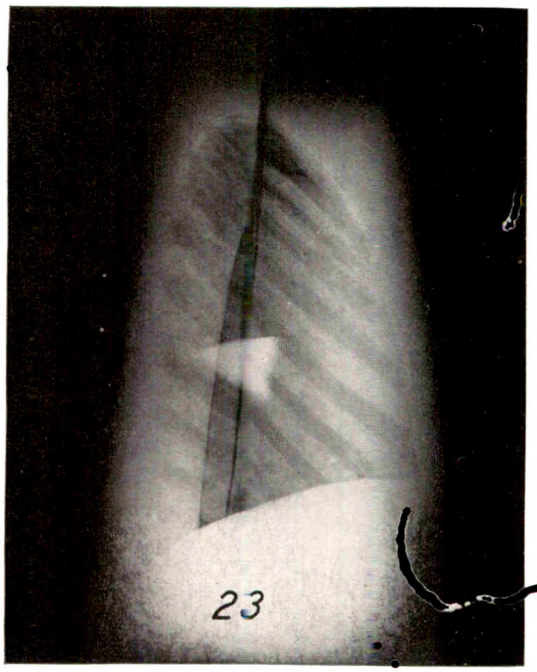


FIG. 22 and 23. A right antero-oblique (Fig. 22) and a right lateral (Fig. 23) roentgenogram of the same condition shown in Figure 21. In the oblique position the shape of the "lesion" has not altered but the change in the position demonstrates clearly that the area involved must be in the region of the dorsal portion of the middle lobe. The lateral view shows the complete involvement of the dorsal tip of the middle lobe instead of just the interlobar fissure. The purpose of this demonstration is to emphasize the fact that a localized pneumonia in this region can cast a shadow indistinguishable from an encapsulated interlobar effusion.

terior of the thorax. With the lung casts inside the mold of the thorax a cast of the mediastinal contents was obtained. A roentgenogram of the chest from a person corresponding in size and weight to the cadaver from which the lungs were obtained was placed interiorly over the anterior and posterior portions of the thorax mold and the position of the ribs was transferred. Since the roentgenogram used was taken at full inspiration it is probable that the lung fissures in the model are a trifle high in relation to the ribs, for the model was made from lungs judged to be in a position of normal breathing. The ribs were then painted on the inner surface of the artificial thoracic cage with white lead. With this variety of molds and casts it was possible to arrange any combination of conditions desired, and once the dummy was assembled roentgenograms could be obtained from any desired position in a manner similar to the taking of roentgenograms of the human thorax.

To demonstrate the position, size and shape of the interlobar fissures thin sheets of lead foil were placed between the lobes, and to accentuate the size and position of the lobes and of the mediastinal contents the visceral pleural surfaces of these structures were covered with thin lead foil. Pathological processes known to exist in human lungs could be simulated by using substances of greater or lesser resistance to roentgen rays than the remainder of the lung casts would possess, thus controlling gradations in density. While it is admitted that this type of model contains a good deal of artificiality, it is possible to prepare an "artificial set-up" and to test the ability of the roentgenologist to accurately interpret the size, shape and location of "lesions" placed at will within the casts of the lungs. Likewise, such a procedure can serve

to assess the accuracy of serial sections by planigraphy.

The size and exact shape of the lungs and of the thorax may not be entirely correct, but they are well within the range of variations that are encountered in human thoraces. The position and contour of the interlobar fissures are correct for the pair of lungs used. The absence of the structures exterior to the chest cavity, of the heart and other tissues of the mediastinum, of the bronchial tree, and of the normal lung "markings" is to be regretted, but it helps to emphasize the position, site and size of such things as the interlobar fissures, the lobes of the lungs and the mediastinal contents.

The main interest to those who are concerned with the interpretation of pulmonary shadows as seen in roentgenograms is, of course, the illustrations which accompany this paper. They are all prints reduced from the actual roentgenograms of the model which were taken on the usual 14×17 inch film. The roentgenograms were taken at a distance of 5 feet from the object with a technique found by trial to give a penetration that would clearly demon-

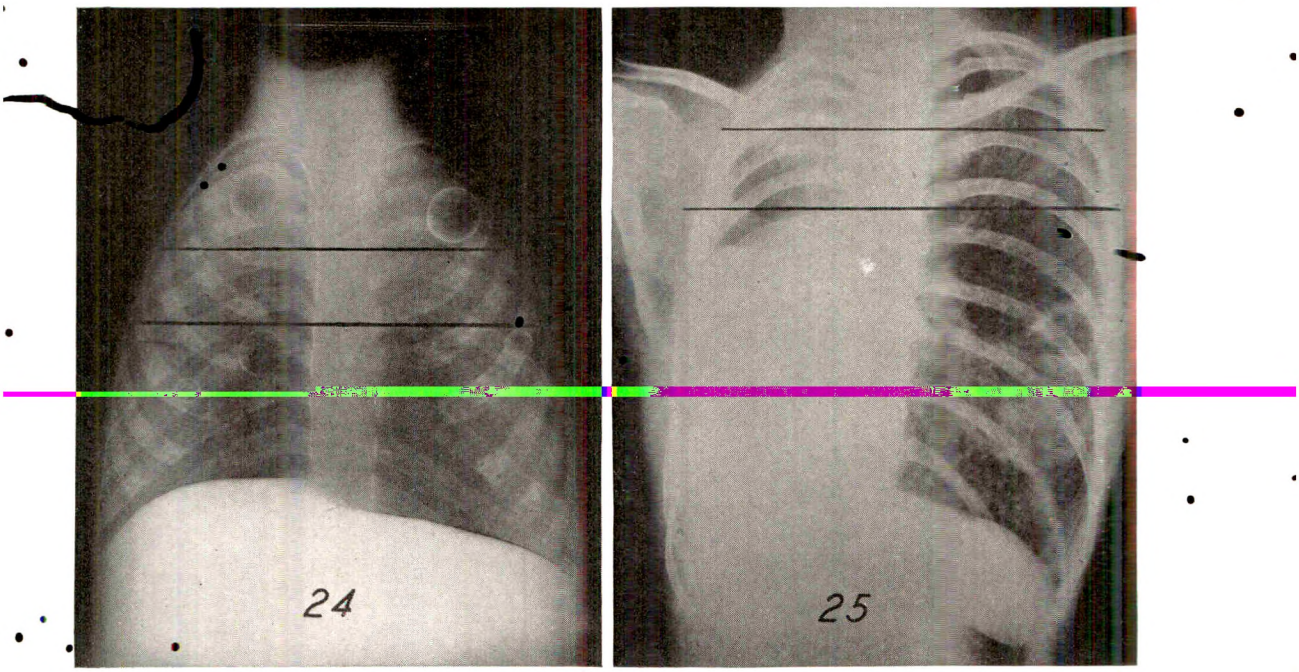


Fig. 24. A posteroanterior roentgenogram with simulated tuberculous cavities placed in the various lobes of the lungs in locations where such lesions are commonly encountered in each lobe. These cavities may vary in their location in all directions but they are preponderantly in the upper portions of each lobe. Since the interlobar fissures are not visible it is not possible to state the location of the three lower cavities with respect to the lobe involved. The horizontal planes, represented by lines, are drawn at the level of the fourth and sixth costovertebral junctions. To appreciate the relation of the interlobar fissures to these lines refer back to Figure 3. A lateral view would show that all but the lowest cavity on the right were located dorsally.

Fig. 25. A posteroanterior roentgenogram of proved tuberculosis taken one year before death. The considerable displacement of the tissue to the right caused the development of an emphysematous left lung. With such a condition a downward displacement of the interlobar fissure would be anticipated. Since the cavity on the left is below the plane drawn at the level of the sixth costovertebral junction and was known to be dorsal in position the location could be stated to be in the upper portion of the left lower lobe. Necropsy revealed a markedly emphysematous lung with a thin-walled cavity in the lateral area of the upper portion of the lower lobe with the upper edge of the cavity 2.5 cm. below the apex of the lobe.

strate the structures within the "dummy." The reader is asked to overlook the aberrant shadows in the roentgenograms and to consider only the main features portrayed, for we admit considerable ineptitude in the artistic handling of the subject matter. The descriptions of the roentgenograms accompany the illustrations.

DISCUSSION

In presenting the studies we have conducted, our main purpose is to give to those concerned with the interpretation and treatment of pulmonary disease a clear and useful concept of the anatomical relationship of the pulmonary lobes and of the interlobar fissures to the bony landmarks as seen in roentgenograms of the chest. The usual posteroanterior, oblique and lateral positions have been illustrated and stereoroentgenograms would have been added had the expense of illustration not been prohibitive. It is hoped that the illustrations reproduced will convey the three dimensional structures of the chest.

It is fully realized that the exact relation-

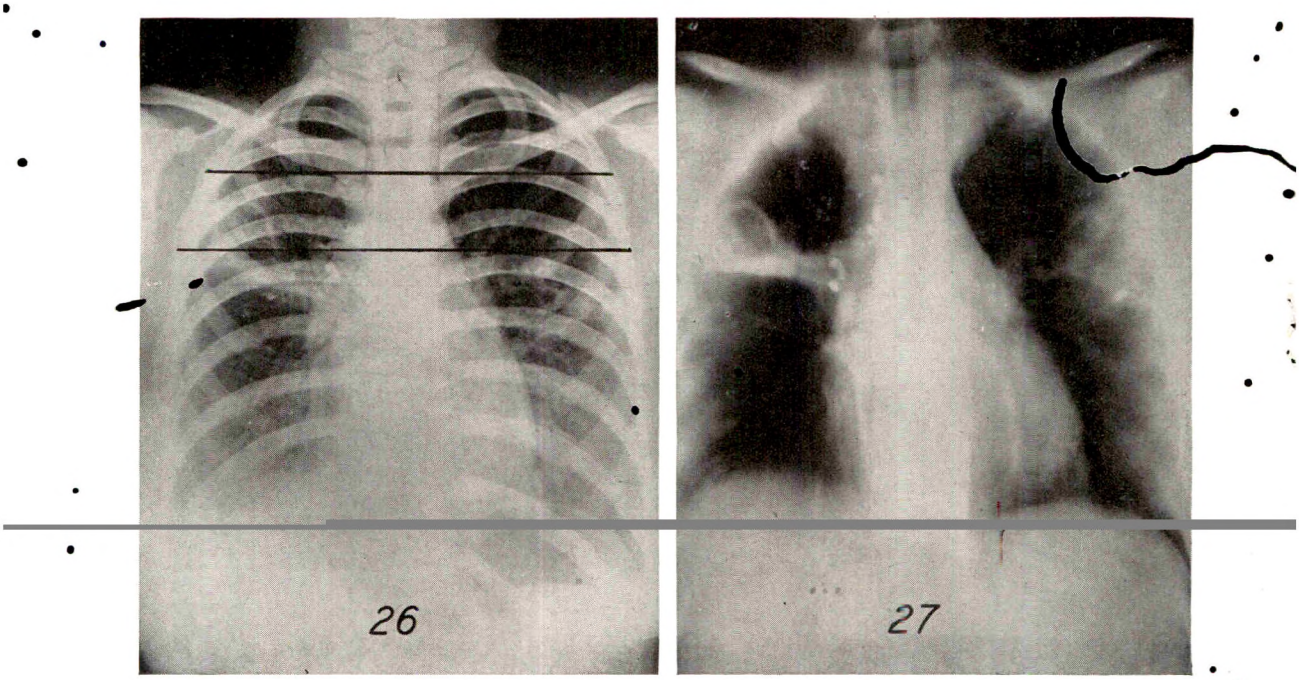


FIG. 26. A posteroanterior roentgenogram of a proved tuberculous case taken about one year before death. There is a pathologic process between the two planes on both sides with a definite cavity cut across by the lower plane on the right. Without the dorsoventral location of the lesion it is impossible to define the lobe involved. Fig. 27. A planigram taken 9 cm. from the table top showing the sharpest outline of the cavity on the right. The lesion on this side was evident at 5 cm. so that it was distinctly dorsal in position. The lesion on the left became evident at 9 cm. and a multilocular cavity was shown ventral to this section. Applying the knowledge gained from studies of the dummy it can be stated that the lesion on the right is in the apical portion of the lower lobe and, on the left, in the upper lobe ventral to the upper portion of the lower lobe. These deductions were proved correct at necropsy. On the right, dense adhesions were present over the area involved and this condition undoubtedly displaced the lesion somewhat upward and laterally. This displacement was not sufficient, however, to invalidate the deductions made. From the lower shelf-like edge shown in Figure 27 one might be led to believe that this represented the interlobar fissure and since the cavity lies above the shelf the pathologic process must be in the upper lobe. Necropsy showed very little involvement of the interlobar fissure while the entire apical portion of the lower lobe was involved. The dense area seen simply represents parenchymal tuberculosis. The course of the interlobar fissure in this region is such that a broad plaque rather than a shelf-like appearance would be expected if the fissure was involved to a degree sufficient to cast a shadow.

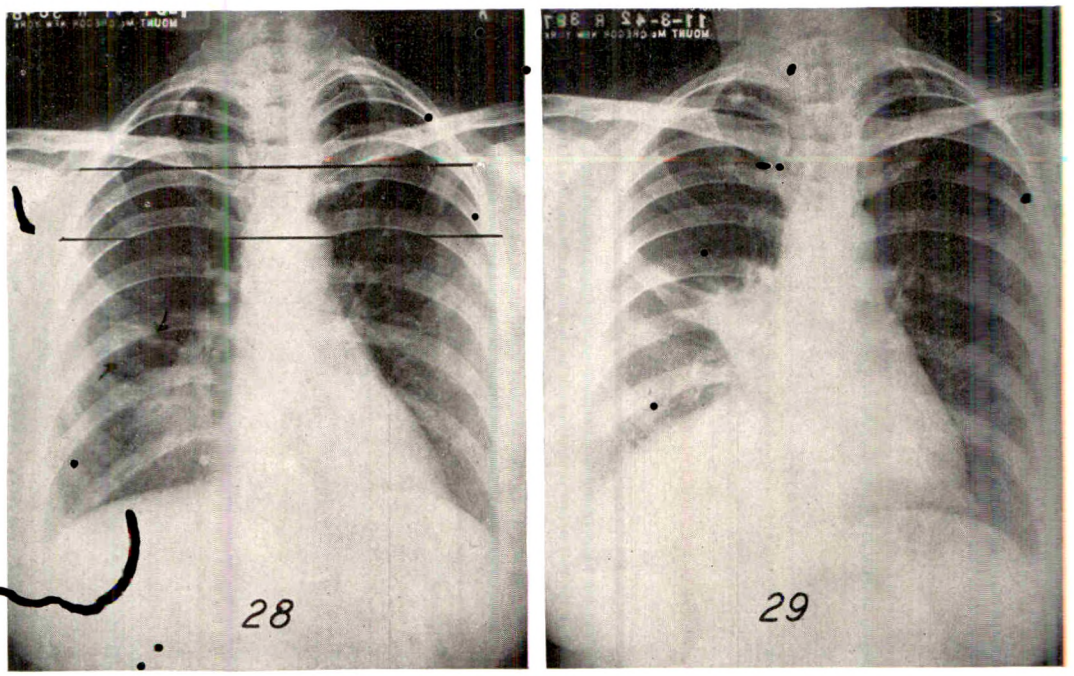
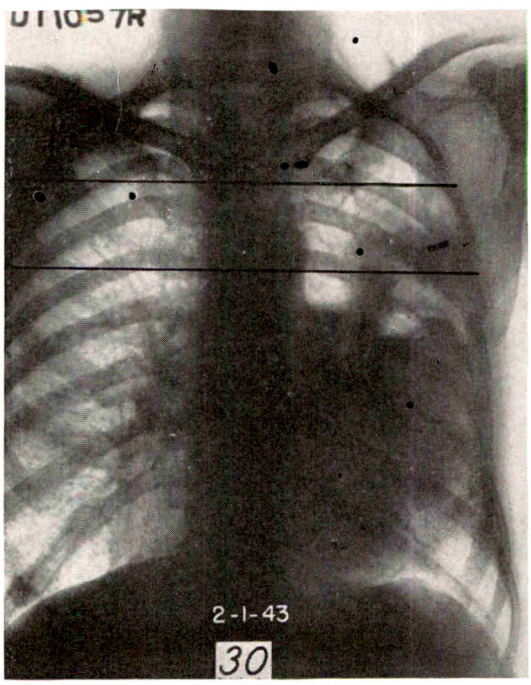
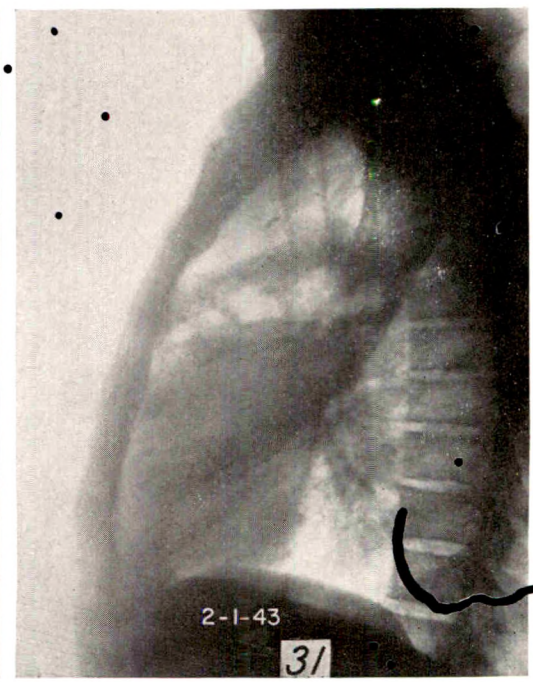


FIG. 28. A posteroanterior roentgenogram of a tuberculous case showing a pathologic process below the lower plane with a definite cavity on the right between the two arrows. This lesion was definitely ventral in position and a diagnosis of middle lobe tuberculosis was made and substantiated when a lobectomy was performed. The tuberculous involvement was found to be in the upper portion of the lobe. Note that the lowest cavity on the right in Figure 24 is similar in position to the cavity present in this case. Fig. 29. A posteroanterior roentgenogram of the same individual three months after operation. This shows the absence of the lesion demonstrated in Figure 28.

ship of the large anatomical units of the lungs to the ribs is not constant, for in both inspiration and expiration the movement of the bony cage and of the pulmonary tissue is in opposite direction. Local pleural adhesions, atelectasis, emphysema, and consolidation of tissue as in pneumonia, also affect this relationship to some extent. An anomaly such as a single lobe in the left lung, for instance, would invalidate any interpretation on the left side. We believe, however, that the relationships observed in the artificial thorax are applicable to interpretations of the average human chest provided there is no undue distortion caused by a considerable amount of pulmonary disease. A practical application of the knowledge gained from studies of roentgenograms of the artificial thorax has been made on 3 cases in which the site of tuberculous cavities was in the less common location and in which proof of the anatomical location was available from necropsy or surgical reports. The deductions made from roentgenograms taken several months prior to necropsy or surgical reports were found to be valid in spite of distortions caused by the pathological condition present at the time the roentgenograms were taken. Horizontal planes through the chest at the level of the fourth and sixth costovertebral junctions have a practical value when interpretations in relation to pulmonary lobes are under consideration. The costovertebral junctions were selected because they are stationary and because the relationship of the lobes and interlobar fissures to the costovertebral junction changes much less than it does to the ventral portions of the ribs during the process of respiration. The practical value of interpretations of chest roentgenograms will be





FIGS. 30 and 31. A posteroanterior and a left lateral roentgenogram, respectively, from a case kindly lent by Dr. J. Burns Amberson of Bellevue Hospital, New York City. The patient was a white male, aged fifty-five, who had had a chill with accompanying cough and continuous sharp pain in the upper anterior portion of his left chest five days previous to hospitalization. A diagnosis of pneumonia was made. Within a few days it became evident that a necrotizing process was present and that a sequestrum of lung was forming. These roentgenograms were taken shortly before surgery was resorted to. At operation a sequestrum the size of a lemon was found in the mid-portion of the left upper lobe, with the pathologic process limited to this lobe. The patient made satisfactory progress following operation.

The illustrations are given to demonstrate how a pathologic process limited to one lobe may indicate the position of an interlobar fissure. That the upper lobe is involved is suggested in Figure 30 from the triangular clear zone resting on the diaphragm (refer to Fig. 3). The lateral view not only confirms the upper lobe involvement but also indicates the position of the interlobar fissure by the sharp limitation of the lesion. That only the upper half of the fissure is outlined can be surmised from a comparison with Figure 13. It is doubtful if the lower ventral angulation of the dark shadow represents the course of the fissure. The presence of a sequestrum is strongly indicated in the lateral roentgenogram.

Horizontal planes through the chest at the level of the fourth and the sixth costovertebral junctions show that a pathologic process is present in this region and that an apical involvement of the lower left lobe has to be ruled out. The lateral view shows a normal apex of the lower lobe and the lesion in the upper lobe, ventral to the lower lobe. The position of the left interlobar fissure corresponds closely to the position shown in the artificial thorax (Fig. 13.)

enhanced provided the following suggestions in relation to the two planes just mentioned are used:

1. Pathologic processes above a horizontal plane at the level of the fourth costovertebral junction will be in the upper portions of the upper lobes. In tuberculosis the majority of lesions will be dorsal in position.

2. Below a horizontal plane at the level of the sixth costovertebral junction pathologic processes in the dorsal half of the chest will be in the lower lobes, especially if the main lesion abuts the dorsal surface of the chest cavity. Pathologic processes in the ventral half will be in the upper lobe on the left and in the middle lobe on the right.

3. The area between horizontal planes at the fourth and sixth costovertebral junctions represents the common variations of the upper edge of the main fissures on either side. In the pair of lungs we have used the upper edge of these fissures is about median in position and probably represents the average. If in this area the pathologic condition is present in the ventral two-thirds of the chest the upper lobes are surely involved. If, however, the lesion adjoins the dorsal surface, involvement of the apex of the lower lobe must be seriously considered. To determine which lobe may be involved pleural endoscopy may have to be employed, especially if the lesion lies in the upper portion of this area.

It is our hope that the data presented may prove to be of practical value to all who are concerned with the interpretations of roentgenograms of the chest and with the treatment of pulmonary disease.

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THE ROENTGENOGRAPHIC MANIFESTATIONS OF ATYPICAL PNEUMONIA OF UNKNOWN ETIOLOGY

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ROYAL CANADIAN NAVAL VOLUNTEER RESERVE

DURING the past decade a moderately infectious constitutional disease with frequent evidence of atypical pneumonia has been increasingly recognized and reported. Dingle and Finland[3] have contributed the most exhaustive recent review of the subject. They state that the condition was recognized clinically as long ago as 1872, and a pathological process was described at about that time. It seems likely that the disease was prevalent before the pandemic of influenza in 1918 and that it existed in the military forces, where it was given such names as "catarrhal fever" and "soldier's pneumonia."

The increased use of roentgenological examination in the diagnosis of inflammatory diseases of the respiratory tract has shown that a variety of roentgenological opacities in the lung fields, often transitory, may occur in the course of such diseases without the grave symptoms associated with the generally recognized forms of pneumonia.[6]

The condition has been graced with numerous terms, the multiplicity of which is due to various investigators' conception of the disease in which the clinical, laboratory and roentgen findings do not follow a definite and clear-cut picture. It has been variously referred to as virus pneumonia, acute influenzal pneumonia, acute pneumonitis, acute interstitial pneumonitis, pneumonitis, atypical pneumonia with leukopenia, current bronchopneumonia of unusual character and undetermined etiology, bronchopneumonia of unknown etiology. Clinically it is probably best termed "primary atypical pneumonia, etiology unknown."[3]

From a roentgenologic standpoint, only an anatomical diagnosis can be given in cases of pneumonia, e.g., "pneumonia of lobar distribution" or "pneumonia of lobular or segmental distribution." Ramsay and Scadding[6] have pointed out that, in order to make a complete diagnosis, both the etiological and anatomical terms must be included in the description of a pneumonia; for example, "pneumococcus type 1 right lower lobar pneumonia" is a specific diagnosis.

According to the roentgenographic shadows produced by the pulmonary lesions, several have reported findings which on analysis of illustrations and descriptions seem to be, for the most part, similar, if not identical.

Seeds and Mazer[10] thus segregate the lesions into three main groups, viz.: "cotton wool," "pseudofibrosis," "wire glass." While these terms are useful in describing a given lesion, they should not be used in identifying a type of pneumonia.

Bowen[2] has reported a group of cases occurring among soldiers in Honolulu and he points out the similarity in roentgenographic appearance to such conditions as pulmonary tuberculosis, lung abscess, acute bronchiectasis, bronchogenic carcinoma and metastatic carcinoma.

Ackermann[1] has also drawn attention to the resemblance of certain cases to pulmonary tuberculosis. However, Ramsay and Scadding state that the roentgenogram is never sufficiently characteristic to justify a dogmatic diagnosis of pulmonary tuberculosis without supportive evidence and without a period of observation.

Kornblum and Reimann[4] have studied a group of cases occurring among medical students, interns and nurses on whom roentgenograms of the chest were obtained at frequent intervals during a mild epi-

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demic of atypical pneumonia. They describe the picture of "acute tracheobronchitis" which they believe represents an early stage of this infectious process. While they recognize the difficulty in making such a diagnosis, they state that the process causes increase in density and size of the hilum and trunk shadows which are ill defined and distinctly blurred, with occasional mottling of the pulmonary tissues about the hilar area. They attach considerable importance to a generalized haziness found throughout the lung field which they consider due to acute congestion of the interstitial blood vessels.

The observations of Ramsay and Scadding suggest that the association of lobar atelectasis with catarrhal infections of the respiratory tract producing no special acute symptoms is more frequent than is commonly recognized. Their report includes 4 cases in which there was evidence of lobar atelectasis and 21 cases of pneumonia. Reimann's[7] Case 9 showed evidence of basal atelectasis. Scadding[8,9] in further reports classifies the pneumonic lesions in two main groups, viz., "benign circumscribed pneumonia" and "disseminated focal pneumonia." The benign circumscribed variety he describes as a fairly well localized, but not sharply defined lesion of uniform density, located usually in the lower lobes. The disseminated focal type produces the roentgenological picture of diffuse, rather coarse mottling in foci varying from 2 to 5 mm. in diameter. He[8] classifies his cases into those with constitutional symptoms but without clinical or roentgen evidence of pneumonia; those with clinical localizing signs (suppressed breathing and fine moist râles); and finally those with both clinical and roentgen localizing signs. He observed a definite lag of two to three days between clinical and roentgen localizing signs.

Briefly, the condition is recognized clinically by fever, cough, malaise, headache, chilliness. Sputum when present is usually mucopurulent, occasionally blood streaked, and rarely frank blood. The physical findings include pharyngeal injection; some cases show prostration and rarely dyspnea with cyanosis. Early pulmonary signs, if present, are limited to fine or medium moist râles with slight dullness. Later, as resolution occurs, loud râles and rhonchi are heard. The leukocyte count is either slightly elevated, normal or slightly depressed. The sedimentation rate may be elevated. The sulfonamides are ineffectual. Complications are few.

Etiology. Dingle and Finland state that the etiology of atypical pneumonia has not been established satisfactorily. The above clinical syndrome may be produced by a number of known bacteria and viruses, and yet in the majority of outbreaks, in which studies were made, none of these agents were identified with the disease.

Kornblum and Reimann hold the same view and remark that the similarity of this disease to other infections of the lungs gave rise to the thought that their epidemic was due to a filterable virus.

Pathology. Cases coming to necropsy are few. According to Dingle and Finland the chief pathological features of fatal cases reported by Kneeland and Smetena and by Longcope are those of a patchy, hemorrhagic interstitial pneumonia associated with acute bronchitis and bronchiolitis. Grossly there are areas of atelectasis and emphysema, small areas of red or gray consolidation, which in some places appear to have become confluent. The bronchi are filled with mucoid or purulent exudate. The microscopic findings include hemorrhagic or mononuclear alveolar exudate and infiltration of alveolar septa with mononuclear cells; the septa may later show thickening with reduction of the alveolar spaces. The walls of the bronchi and trachea show necrosis and infiltration with polymorphonuclear cells.

REPORT OF CASES

This report is based on 178 cases of bronchopneumonia, demonstrated by roentgenographic examination, occurring in personnel of the Royal Canadian Navy who were referred with clinical features con-

forming to those described. Bacteriological studies, when done, revealed no positive pathogens. This group had, as do all R.C.N. personnel, routine chest roentgenograms on entry.

Method. As a routine, posteroanterior and the appropriate lateral roentgenograms of the chest are obtained either at the bedside or in the roentgenographic department, depending on the condition of the patient. The majority of cases in this group include admission and progress roentgenograms. A case is not released from the hospital until the chest roentgenogram shows complete clearing of the process.

ROENTGENOGRAPHIC FINDINGS

There is no notable relationship between the anatomical extent of the disease as shown in the roentgenogram and the severity of the symptoms. Pneumonic infiltrations have been discovered in the enlistment roentgenograms of 12 recruits who have had no complaints other than a recent "chest cold." The appearance and anatomical extent of these infiltrations are identical with those of individuals in the hospital who were considered too sick to be transported to and from the department and on whom roentgenograms secured at the bedside were necessary. A lag of two to three days has occasionally been noted between the clinical and roentgen evidence of pneumonia.

TABLE I

FREQUENCY OF LOCATION OF PNEUMONIC INFILTRATIONS

	per cent
Left lower lobe	44
Right lower lobe	35
Right upper lobe	7
Accessory right lower lobe	4
Left hilar	4
Right hilar	2
Left upper lobe	2
Right middle lobe	2

Location. The frequency of involvement of the various lobes is given in Table I. One hundred and eighty-one lobes were involved in 178 cases. Eighty per cent of lesions were confined to the lower lobes.

Roentgenographic Appearance. In almost all cases, the parenchymal infiltration manifests itself by a peribronchial distribution, viz., bronchopneumonia. While a lesion in the posteroanterior projection may appear of lobar distribution, nearly always the lateral projection reveals the process to be a segmental, confluent bronchopneumonia, radiating in a fanwise manner from the hilum. It is true that an infiltration of lobar distribution is occasionally demonstrated in a case with a clinical picture conforming to that described.

Early infiltration commences in one of two ways:

First, an ill defined, blurred, localized structural accentuation occurs. If the disease progresses anatomically, the peribronchial blurring extends and coalesces, producing a frank parenchymal infiltration (Fig. 1). This varies in density from a slightly impaired translucency to a ground glass appearance and occasionally progresses to a moderate density, but rarely equals that of the cardiac shadow unless complicated by pleuritis (Fig. 2). An infiltration of moderate density may be visualized, for example, in the posteroanterior view, but this has been shown to be almost always transparent in the lateral projection. Accentuated structural markings are usually visible through this density and appear prominent.

The second method of development is first manifested by an area of localized, rather dense structural accentuation. If the anatomical extent progresses, flocculent densities, peribronchial in distribution, appear (Fig. 3).

Finally, occasionally both homogeneous and flocculent densities are demonstrated in the same lesion, thereby resulting in a homogeneous lesion through which "double densities" are visualized which merge imperceptibly into the adjacent infiltration.

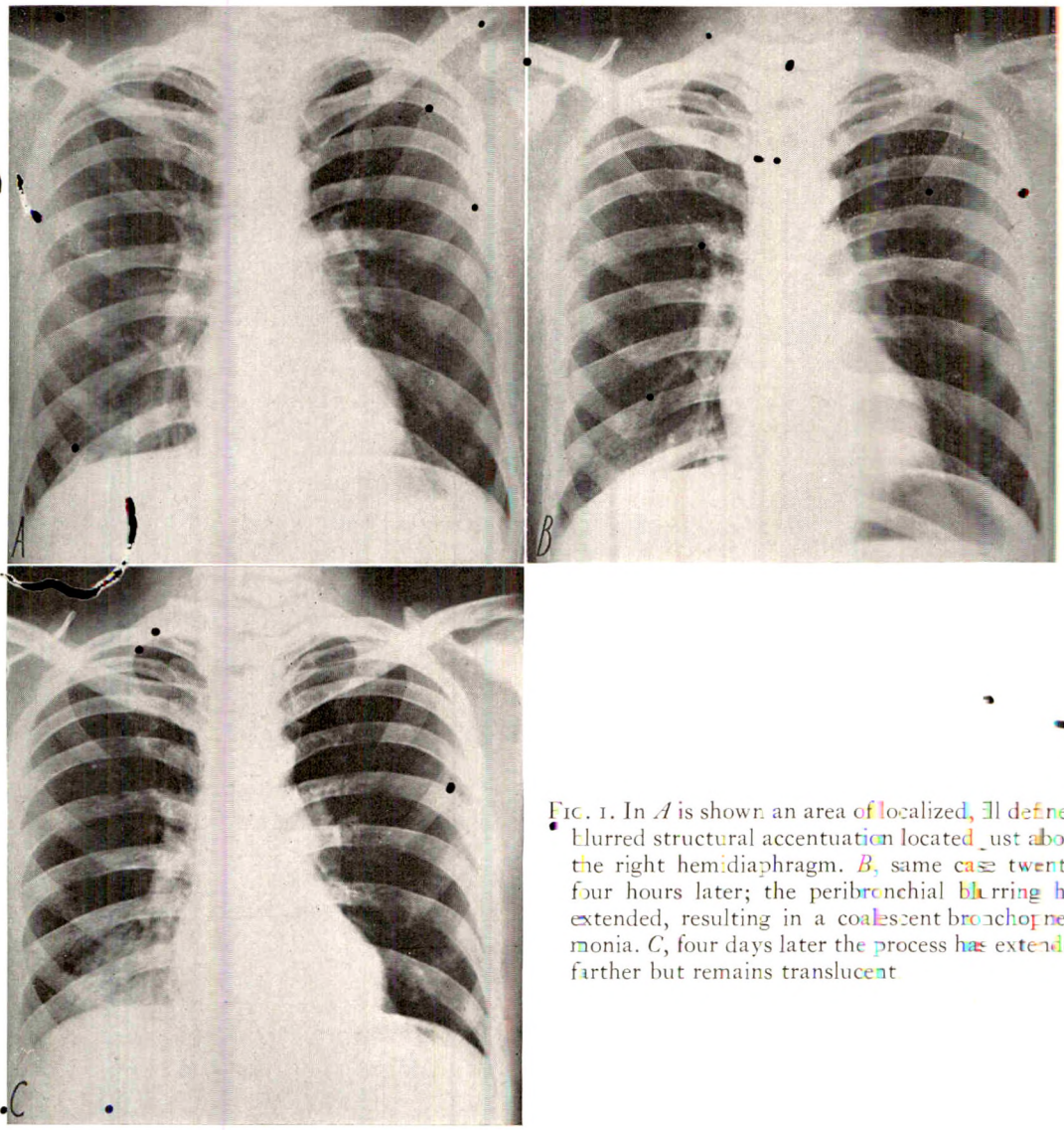


Fig. 1. In *A* is shown an area of localized, ill defined, blurred structural accentuation located just above the right hemidiaphragm. *B*, same case twenty-four hours later; the peribronchial blurring has extended, resulting in a coalescent bronchopneumonia. *C*, four days later the process has extended farther but remains translucent.

To summarize:

A. Homogeneous Group:

1. The purely homogeneous lesion as described above.

2. The ill defined and blurred structural accentuation—probably an early manifestation.

3. The "cotton wool" lesion, composed of several adjacent homogeneous circular lesions which do not coalesce.

B. *Flocculent Group:*

1. The purely flocculent and rather dense lesion.

2. The dense linear accentuation—probably an early manifestation.

3. Linear accentuation with occasional areas of dense flocculent infiltration.

C. *Homogeneous Flocculent Group:*

The frequency of these various types has been analyzed and is given in Table II.

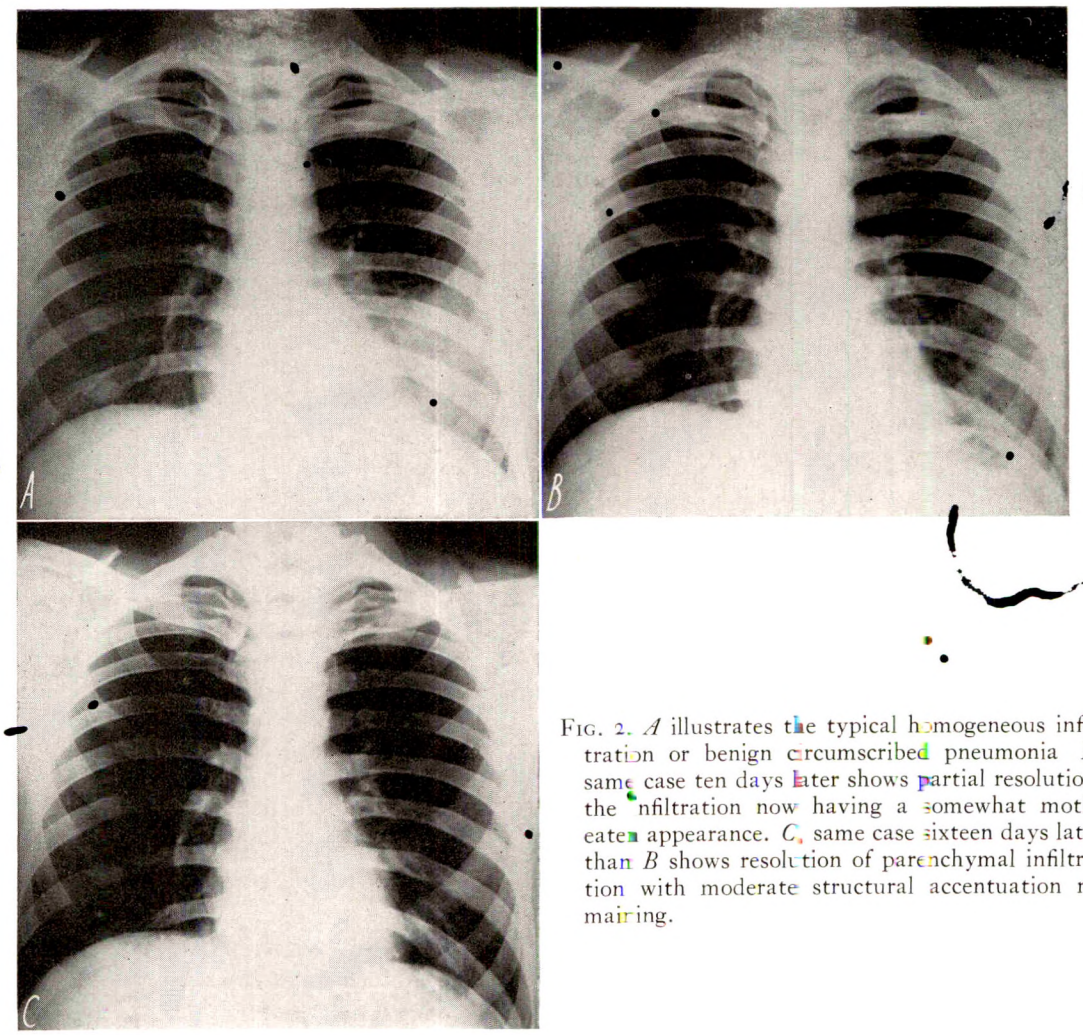


FIG. 2. *A* illustrates the typical homogeneous infiltration or benign circumscribed pneumonia *B*, same case ten days later shows partial resolution, the infiltration now having a somewhat moth-eaten appearance. *C*, same case sixteen days later than *B* shows resolution of parenchymal infiltration with moderate structural accentuation remaining.

Hilar adenopathy, increase in density of the hilum, ill defined accentuation of the trunk shadows with obliteration of the translucent space between the inner border of the right hilum and the right border of the heart have been demonstrated in a number of basal infiltrations.

Resolution in the homogeneous group is revealed by a reduction in total density at which time the infiltration assumes a moth-

TABLE II

FREQUENCY OF THE TYPES OF ROENTGENOGRAPHIC DENSITIES

Homogeneous	Homogeneous with Structural Accentuation	Cotton Wool	Flocculent	Flocculent with Structural Accentuation	Dense Structural Accentuation	Homogeneous Flocculent
30.8%	12.3%	3.3%	20.7%	12.3%	6.7%	13.9%
	46.4%			39.7%		13.9%

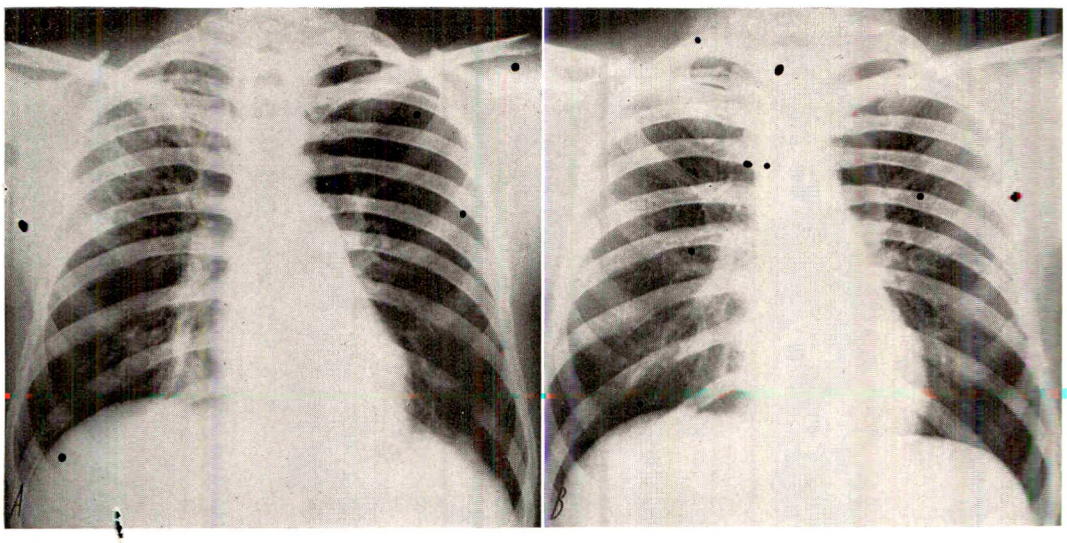


FIG. 3. *A*, an example of the flocculent or disseminated focal infiltration occurring at the right apex, resembling pulmonary tuberculosis; parenchymal infiltration is also demonstrated at the right cardiohepatic angle. *B*, same case eighteen days later showing complete resolution.

eaten appearance. When regression of the infiltration is complete, the structural accentuation often recedes more slowly and is last to disappear. The flocculent lesions and associated structural accentuation resolve more or less together, becoming gradually less dense (Fig. 2).

Certain lesions of either homogeneous or flocculent type, when confined to the upper lung field, resemble pulmonary tuberculosis. Re-check examination in two weeks usually allows a definite opinion (Fig. 3).

Pleuritis with effusion as recognized on the roentgenogram is a rare finding. Evi-

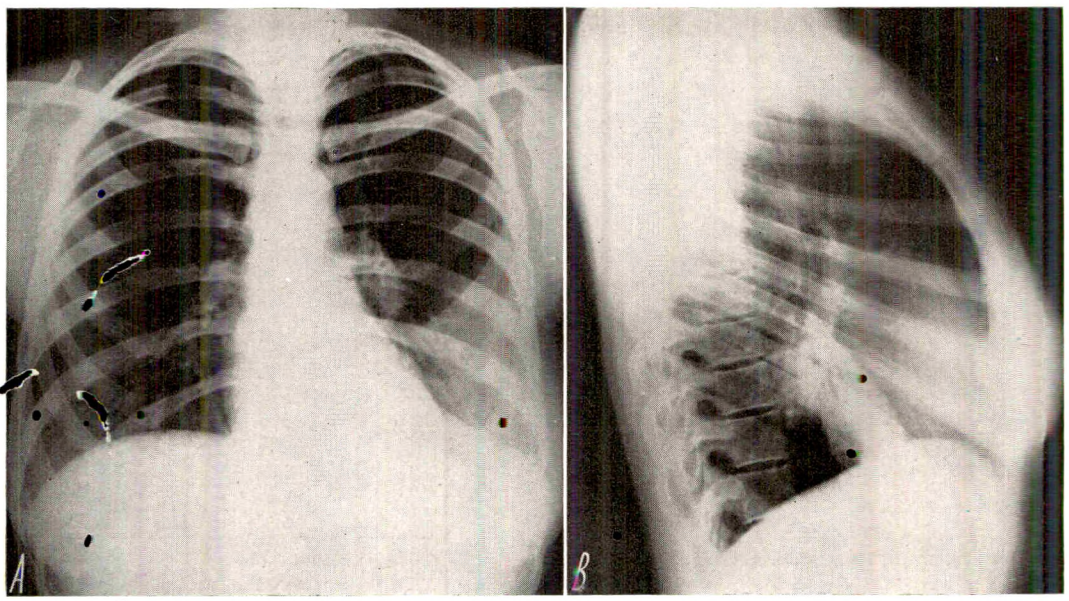


FIG. 4. *A* and *B*, interlobar pleuritis with effusion located at the lower end of the left fissure. Note the similarity between 4*A*, 1*C* and 2*A*; the lateral roentgenogram makes an accurate differentiation.

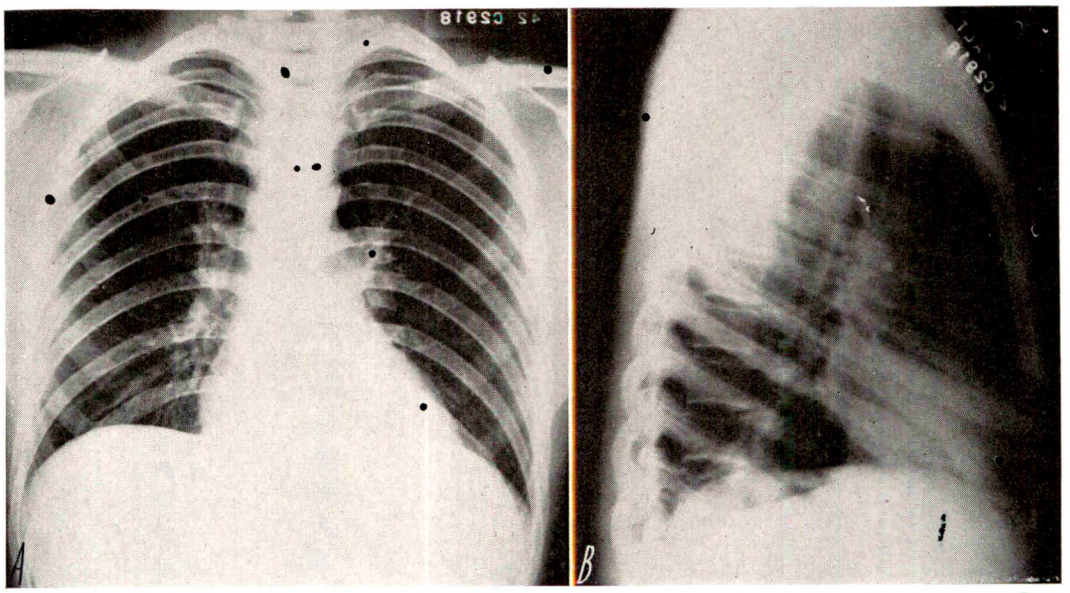


FIG. 5. *A*, portable anteroposterior roentgenogram shows no evidence of parenchymal infiltration, but slight elevation of left hemidiaphragm is present. *B*, the left lateral roentgenogram clearly demonstrates a triangular density radiating downward in a fanwise manner from the left hilum and is confined to the left lower lobe, thus localizing the process behind the heart.

dence of coexistent interlobar or parietovisceral pleural thickening was observed in 22 cases and then only when the parenchymal process was adjacent to the pleura.

The homogeneous density of parenchymal infiltration is similar in appearance in many cases to that of interlobar pleuritis. As described and illustrated by Levitin and Brum,[5] the lateral projection has served to make an accurate differentiation (Fig. 4).

The lateral projection was used in 100 cases in this series. In 25 of these, it was of no assistance; in 7 the appropriate lateral roentgenogram not only localized the process, but provided an insight into the actual area of parenchyma involved. In 3 cases the process was demonstrated either below the summit of the dome or behind the heart and in none of these was the process recognized in the posteroanterior projection (Fig. 5 and 6).

Elevation of a Hemidiaphragm. This finding has been noted in a total of 38 cases, or 21.3 per cent, and has been interpreted as evidence of atelectasis (Fig. 7). This has varied from slight to marked elevation;

TABLE III

LENGTH OF TIME FOR ANATOMICAL RESOLUTION*

Type of Lesion	No. of Cases	Average No. of Days
Homogeneous infiltration	52	16.0
Flocculent infiltration	46	15.4
Combined infiltration	9	13.7
Slight elevation of hemidiaphragm	13	14.6
Moderate and marked elevation of hemidiaphragm	7	17.4

* A total of 142 cases had serial examinations of which 127 are included in Table III and 15 in Table IV.

TABLE IV

CASES DISCHARGED AS CLINICALLY RESOLVED AND IN WHICH ANATOMICAL RESOLUTION WAS NOT ENTIRELY COMPLETE

Lesion	No. of Cases	Average No. of Days
Residual structural accentuation	8	23.5
Residual elevation of hemidiaphragm with or without residual structural accentuation	7	33.5

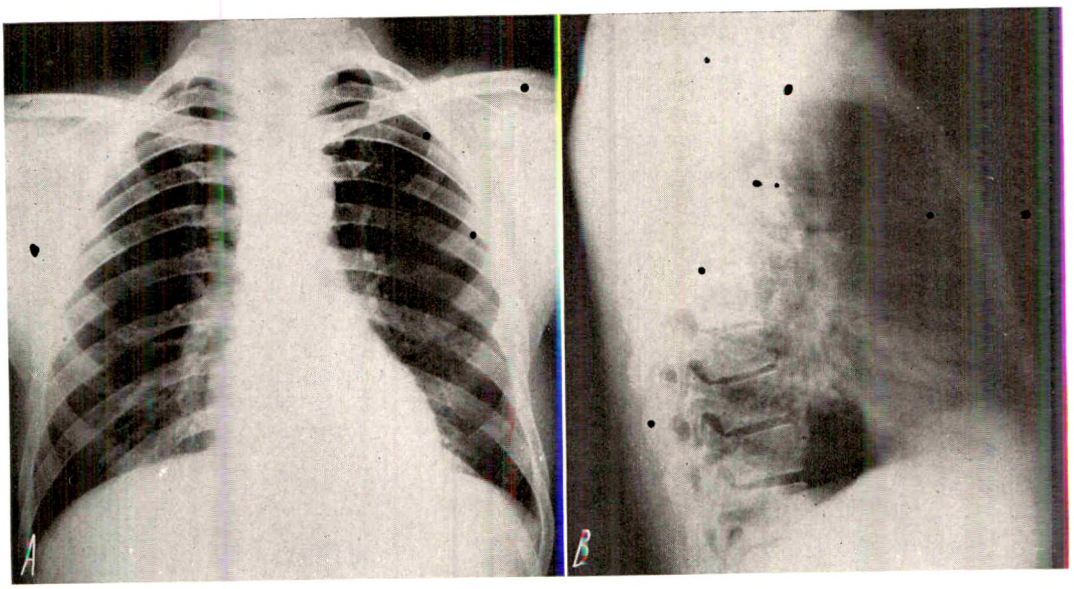


Fig. 6. *A*, posteroanterior roentgenogram shows no evidence of parenchymal infiltration. *B*, right lateral projection (reversed) demonstrates an area of parenchymal infiltration located in the right lower lobe at its posterior costophrenic angle below the summit of the right hemidiaphragm.

Fleischner's lines have also been noted. Only rarely are other signs of atelectasis observed, e.g., mediastinal displacement, narrowing of rib spacing.

As noted in Table III, atelectasis as a coexistent finding results in a prolongation of resolution.

Complications. As shown in Table III, anatomical resolution occurs in approximately two weeks. This is occasionally prolonged, as Table IV shows, but these cases have been clinically clear. Not included in Table IV are 5 cases of non-resolution which developed complications.

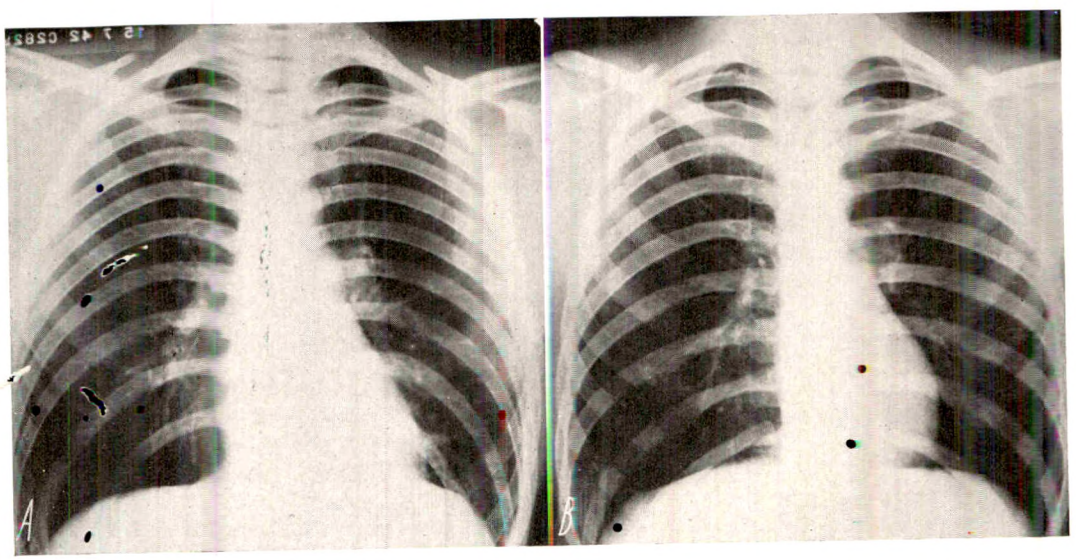
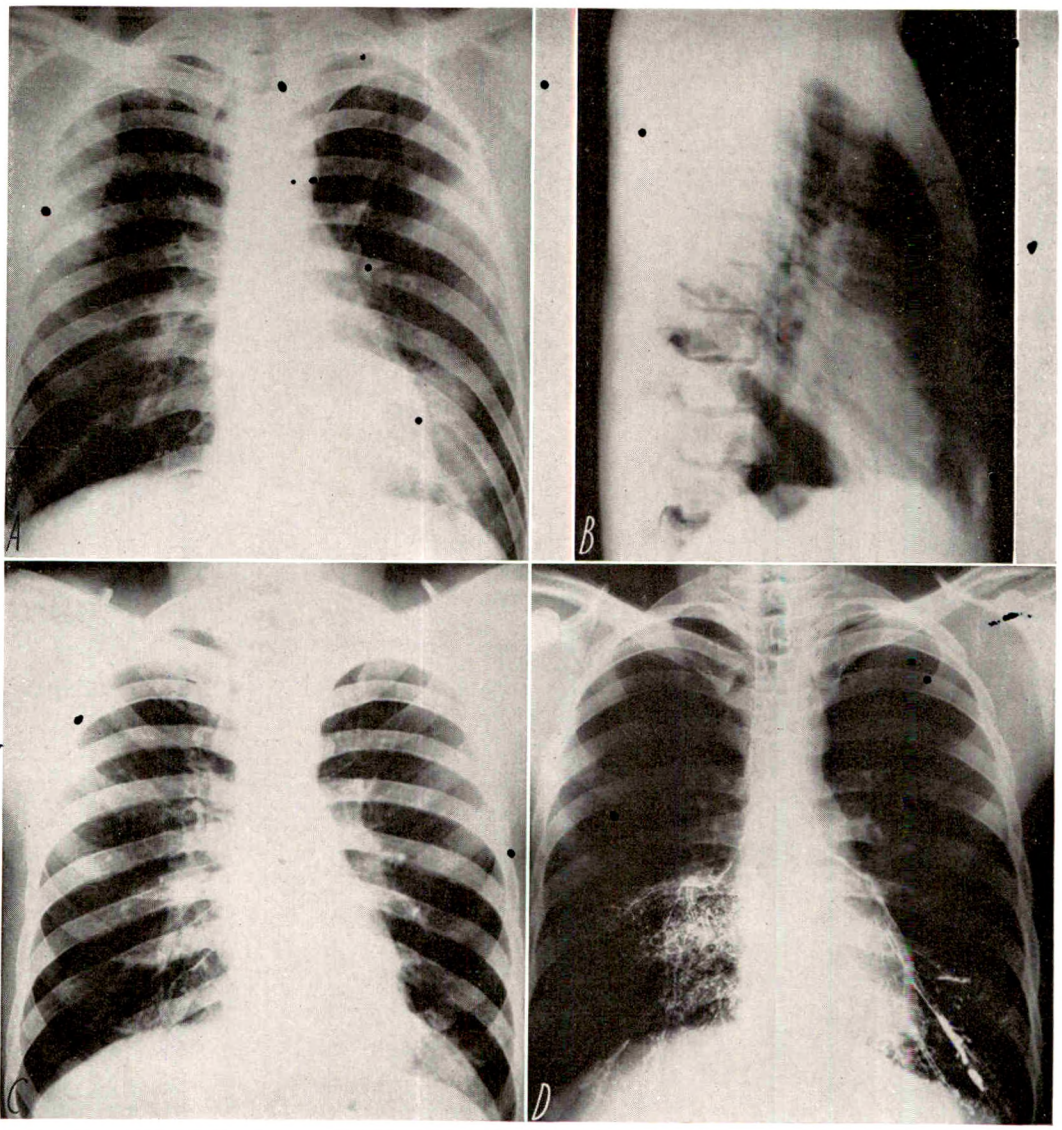


Fig. 7. *A*, portable anteroposterior roentgenogram shows a small patch of parenchymal infiltration at the left base medially and elevation of the left hemidiaphragm. *B*, same case eighteen days later shows resolution of the process and return to normal relationship between the domes of the diaphragm.



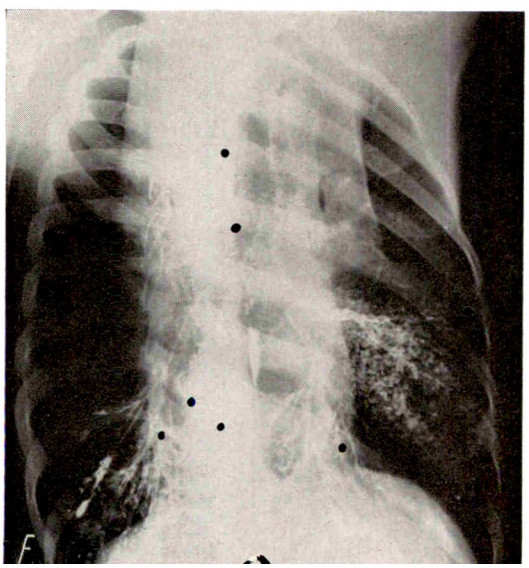


FIG. 8. *A* and *B*, portable anteroposterior and left lateral roentgenograms of chest show an area of homogeneous circumscribed pneumonia at the posterior and basal portions of the left lower lobe. *C*, same case sixty-two days later shows only partial resolution. *D* and *E*, posteroanterior and left oblique bronchograms thirty-nine days later show fusiform dilatation of a bronchus at posterior and basal segment of left lower lobe with failure of smaller bronchi to fill.

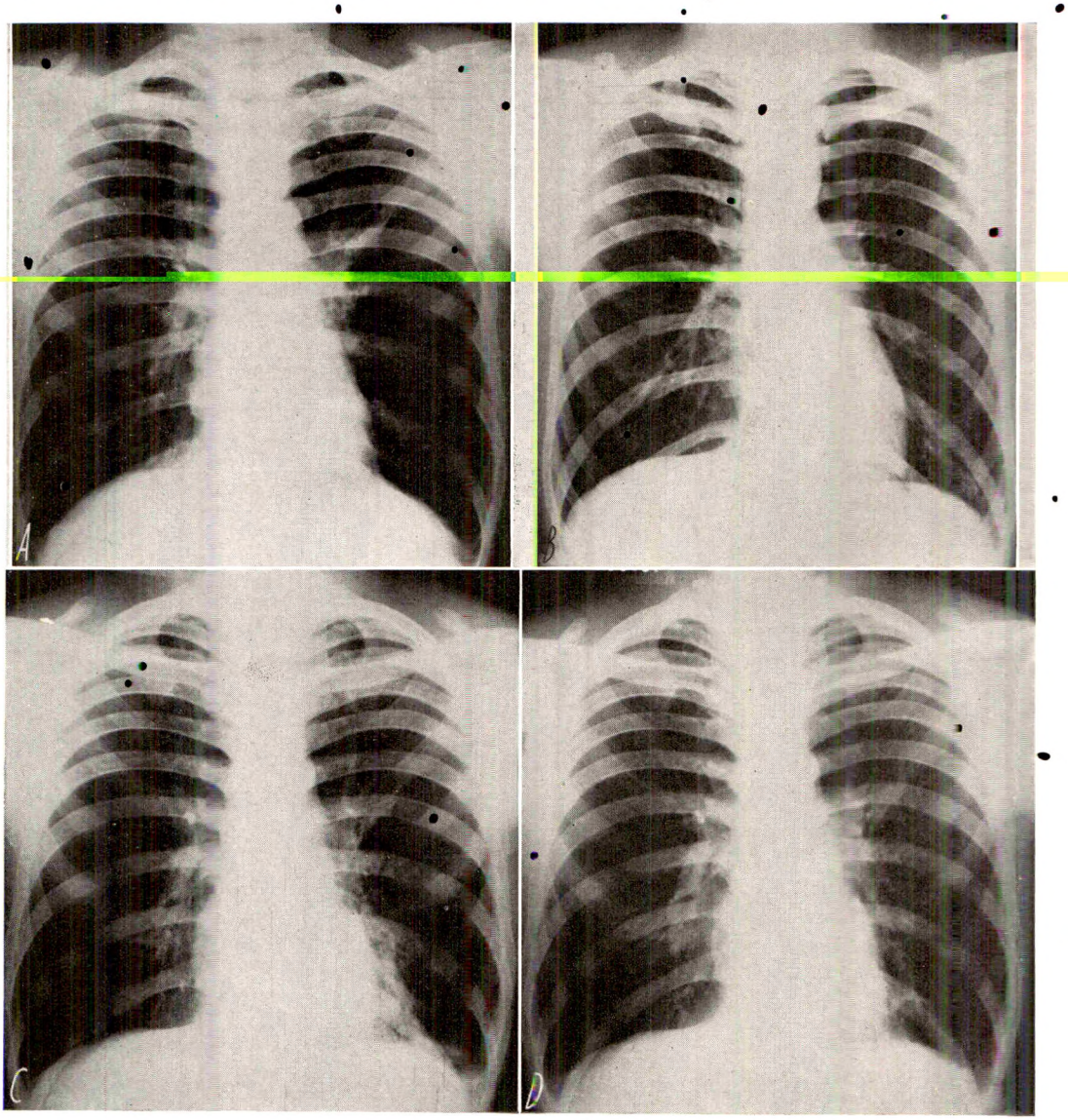


Fig. 9. *A*, evidence of parenchymal infiltration extending in a linear manner up and out from left hilum. *B*, same case twenty-seven days later in which there is demonstrated localized structural accentuation with hazy parenchymal infiltration at left base. *C*, same case ninety-nine days later. Pronounced and dense infiltration at left base; clinical evidence of asthma. *D*, twelve days later, partial resolution of pneumonic process.

Three of these showed clinical evidence of bronchiectasis. Lipiodol bronchograms were done in 2 cases and were confirmatory (Fig. 8). The 2 remaining cases showed roentgen evidence of non-resolution and clinical evidence of asthma (Fig. 9).

DISCUSSION

From the foregoing it is submitted that the condition described is identified with constitutional symptoms with localization in the respiratory tract which frequently advances to parenchymal infiltration. Clinical diagnosis at present should be termed "atypical pneumonia, etiology unknown."

Roentgen examination in positive cases reveals evidence of parenchymal infiltration, the extent of which does not coincide with the severity of the symptoms and is predominantly located in the lower lobes.

This series revealed localization in one lobe in the great majority.

The appearance of the infiltration is either transparent and homogeneous, flocculent or a combination of these, occasionally associated with hilum prominence. It is considered that this classification coincides with Scadding's "benign circumscribed" and "disseminated focal" types and this classification is endorsed.

When located in an upper lung field, the disseminated focal infiltrations resemble pulmonary tuberculosis of the confluent flocculent type and the benign circumscribed consolidation bears a close resemblance to the exudative, homogeneous tuberculous infiltration referred to in the German literature as the Assmann infiltration. A re-check roentgenogram in two weeks usually allows for a definite opinion lacking confirmatory clinical and laboratory data.

The use of the lateral projection has been of value. When one considers the volume of lung parenchyma located below the summit of the diaphragm and behind the heart which is not visible in the posteroanterior projection, the value of the appropriate lateral roentgenogram becomes increasingly apparent. Moreover, this projection provides an insight into the extent of the process and thereby serves to localize the infiltration accurately. Occasionally, consolidation is demonstrated only in this view. Finally, the lateral roentgenogram serves to differentiate the benign circumscribed infiltration from interlobar pleuritis.

Elevation of a hemidiaphragm in conjunction with a basal lesion has been encountered more frequently than in reports of others. While it is recognized that the finding may be due to a lack of elasticity incident to the coexistent inflammatory changes, a lobular or partial atelectasis seems more appropriate. The disease is characterized by a respiratory mucous membrane reaction with exudation in some stages. It is a reasonable conjecture that in a certain number plugging of bronchioles takes place with resulting atelectasis. Evidence of coexisting atelectasis gives rise to an appreciable prolongation in length of anatomical resolution. Further, in a few cases, the combination was followed by evidence of non-resolution and bronchiectasis. The importance of coexisting atelectasis is stressed, for if this becomes irreversible, the development of permanent bronchiectasis may follow.

Resolution, however, is the rule, leaving no sign of the previous infiltration and occurring on the average within two weeks. There are a few cases with roentgen evidence of non-resolution and clinical evidence of asthma. To date there has been no mortality.

Accordingly, the roentgenographic picture, while fairly characteristic, may show sufficient deviation to be confused with that of pneumococcus lobar pneumonia, bacterial bronchopneumonia, tuberculosis, pulmonary abscess, acute bronchiectasis, metastatic infiltration and, of course, the known virus and rickettsial diseases that involve the lung.

SUMMARY AND CONCLUSIONS

1. One hundred and seventy-eight cases of atypical pneumonia, with roentgen evidence of bronchopneumonia, are presented.

2. Scadding's roentgen classification of benign circumscribed and disseminated focal types is endorsed.

3. The occurrence of accompanying atelectasis is reported and its importance discussed.

4. The importance of the appropriate lateral projection of the chest is stressed.

5. Resolution is the rule. Accompanying atelectasis probably accounts for the rare development of bronchiectasis. Non-resolution with clinical evidence of asthma has also been observed.

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A PLEA FOR THE PREVENTION OF BRONCHIECTASIS*

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THE JUSTIFICATION for this presentation is based upon the assumption that the general medical profession is not aware of the prevalence of bronchiectasis, nor does it appreciate the significance of this disease. That a roentgenologist should feel called upon to discuss the subject of the prevention of bronchiectasis is due to the belief that no specialist in the practice of medicine has a better opportunity to view this disease in all its varied aspects. To understand the need and the measures available for the prevention of bronchiectasis, one must have knowledge of the incidence, pathology, pathogenesis, etiology, symptomatology, clinical course, prognosis, economic importance and treatment of this malady. Bronchiectasis cannot be cured *in situ* and since surgical excision of the diseased tissue is limited to selected cases, it is obvious that the attack upon the problem of bronchiectasis lies in prevention rather than cure.

To a large part of the medical profession bronchiectasis is but a name and to a surprising number of physicians, it is a name they do not clearly understand. There is little wonder, then, that the prevalence and clinical significance of this disease is not generally appreciated. With a little thought, one can, I believe, recognize the reason for this state of affairs. If bronchiectasis were a new and startling disease, every practitioner throughout the land would be familiar with and interested in it. But bronchiectasis is an old disease and has, up to the present time, attracted the attention of only specialized medical groups. It has never been popularized as an affection of importance and general clinical interest. First described by Laennec in 1819, it was a disease which, for the next century, largely attracted the attention of pathologists who devoted much time to investigations of its pathology, pathogenesis and etiology. Since the condition was not readily recognized clinically, it attracted but little attention from practitioners, and treatment was a matter to which no thought whatever was given. With the introduction of the bronchoscope by Killian, of Vienna, in the 1880's and its perfection and popularization by Jackson and his disciples in this country during the early part of the present century, the study of the bronchial tree in the living subject became possible and opened up new vistas for clinical investigation. And with the aid of another remarkable and comparatively recent discovery, the roentgen ray, visualization of the bronchial tree by means of the insufflation of bismuth by Jackson in 1918 and later (1922) the introduction of iodized oil by Sicard and Forestier made possible the visualization of the anatomy of the bronchial tree in health and disease. These innovations may rightly be said to mark the real beginning of our understanding of the disease known as bronchiectasis.

There is yet another reason for the lack of recognition on the part of the general medical profession. The fact that the diagnosis of bronchiectasis remains largely in the hands of specialists does not tend to encourage interest on the part of the general practitioner. In the final analysis, a positive diagnosis can be made only by bronchography. This is a procedure that is not available to the average physician and one which is being carried out successfully in only the more modern and well equipped hospitals. With this state of affairs, the average physician thinks of bronchiectasis as a disease of interest only to the specialist such as the bronchoscopist, the radiologist, the internist specializing in chest diseases

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and the thoracic surgeon and thus comes to adopt the attitude that, to him, bronchiectasis is of no immediate concern. However, in this discussion we are not primarily concerned with bronchiectasis but rather with the conditions which may terminate in bronchiectasis. These are the diseases which constitute a large percentage of the work of the general practitioner. The recognition on the part of the general practitioner that many of the conditions which he sees daily are those which may develop into bronchiectasis should give him an immediate and first hand interest in this subject. And when he comes to realize that the prompt recognition and the proper treatment of the diseases which predispose to bronchiectasis are essential to its prevention, he will have come to the realization that while the specialist deals with the end results of this condition and can hope to cure only a small proportion, the key to the problem of prevention is in the hands of the general practitioner.

Why should radiologists concern themselves with the prevention of bronchiectasis and why are they in such an excellent position to view this disease in all its varied aspects? Roentgenography of the chest has become an indispensable procedure in the practice of medicine. Furthermore, its widespread use in survey studies of the chest in industry, in schools, and in the military services foreshadows the day when the yearly roentgen study of the chest of the entire population will be routine. Such surveys bring to light many conditions recognized today as forerunners of bronchiectasis and thus the roentgenologist has the opportunity to detect many individuals who may develop this disease. Bronchiectasis, although more prevalent in adults, is primarily a disease of childhood. The roentgenologist in his daily work with children can detect those conditions which often result in bronchiectasis long before any symptoms of this disease are present. The pediatrician who sees the patient during the developmental stage of bronchiectasis loses sight of the patient as an adult, now a victim of bronchiectasis. The internist may care for a patient during the illness destined to result in bronchiectasis but may not be called upon to see him when bronchiectasis has become established. The phthisiologist may have a patient referred to him for the diagnosis of tuberculosis, but in the absence of evidence of this disease he may or may not recognize the presence of bronchiectasis. The experience of the bronchologist is such that bronchiectasis is always uppermost in his mind. Unfortunately, by the time he is consulted the disease is usually well established and prevention is out of the question. The thoracic surgeon is in much the same position. The general practitioner will see the patient as a child or an adult and will encounter bronchiectasis in all of its stages. It is not, however, easy to connect the symptoms of the adult with full blown bronchiectasis with the relatively mild and inconsequential symptoms of the child with a mild tracheobronchitis following an attack of whooping cough or measles. The roentgenologist, on the other hand, sees the patients of all these specialists and of the general practitioner as well and in his daily work there are usually several patients who represent the various stages in the development of bronchiectasis. There is the child with the annoying cough and chronic nasal discharge; there is the internist's patient just recovering from pneumonia who has persistent definite roentgen changes at the site of the pulmonary involvement after clinical recovery is complete. The chest specialist has requested a roentgenogram of the young man who recently had a slight hemoptysis. Tuberculosis is suspected but the roentgenogram fails to substantiate this diagnosis. There is, however, definite intensification of the trunk shadows at both bases. The bronchoscopist refers a patient who has suffered from a chronic cough for many years. The chest roentgenogram reveals an unsuspected foreign body of the lung. The surgeon has requested a bronchography preliminary to a lobectomy for bronchiectasis. This series of cases, with its many varia-

tions, is a daily and familiar picture to the average roentgenologist. He is constantly confronted by the life cycle of bronchiectasis as a lesion of relatively little importance to the bronchographic picture of large bronchial dilatations and sacculations, and he cannot help but concern himself with its prevention.

To understand the need for the prevention of bronchiectasis one must have knowledge regarding its entire picture from the onset to its final termination and must know just what it means to an individual to have this affliction. A voluminous literature has come into being regarding every aspect of this malady. In digesting this material, one is impressed with the controversy surrounding almost every phase of the condition. There appears to be no unanimity of opinion regarding anything about bronchiectasis with the exception of its definition. All seem agreed that bronchiectasis is nothing more than a condition in which the bronchi show an abnormal dilatation. The extent or character of this dilatation, the amount of the bronchial tree involved, the duration of the condition, the presence or absence of symptoms and the possibility of complications have nothing to do with the definition. It would serve no useful purpose in this discussion to enter into all the controversial aspects of this disease, so an attempt will be made to adhere to more or less well established facts.

INCIDENCE OF BRONCHIECTASIS

Is bronchiectasis sufficiently prevalent and are its harmful effects such as to warrant the interest of every physician?

Bronchiectasis was formerly regarded as a comparatively rare disease. One gains some idea of this by consulting textbooks of pathology and medicine of some twenty to thirty years ago. A few paragraphs, rarely a page or so, was devoted to this condition. Contrast this with the space now devoted to a full discussion of every phase of bronchiectasis in modern textbooks, and the growing importance of this disease becomes readily apparent. This change has been brought about by our modern methods of diagnosis which reveal for the first time the real prevalence of bronchiectasis. And now with the remarkable advances being made in the surgical treatment of bronchiectasis, this disease is rapidly coming into its own.

It is surprising, however, how little is to be found in the literature regarding the incidence of bronchiectasis. Writers on the subject refer to the disease as being very common but apparently there are few statistics to prove this point. Leopold[19] states that the importance of bronchiectasis is attested by the fact that in frequency this disease stands second only to tuberculosis among chronic infections of the lungs. According to Hamman[15] postmortem statistics indicate that bronchiectasis is found in about 1.5 per cent of routine autopsies. Naturally, the diagnosis of bronchiectasis is more common in those clinics in which bronchoscopy is available. It must be remembered also that the published reports constitute, in the main, the more severe cases and that the vast majority of patients with bronchiectasis are dismissed by their physician with a diagnosis of chronic bronchitis, and then there is that even larger group of relatively mild cases who never seek medical attention. From these facts it can be seen that it is probably not possible to obtain a true estimate of the incidence of bronchiectasis.

However, certain facts regarding the incidence of bronchiectasis are fairly well established. While bronchiectasis shows no particular sex predilection, statistical studies indicate that the disease is slightly more common in males than in females. Farrell[10] reported cases in 52 males, 48 females; Perry and King,[20] 214 males, 186 females. This slight male predominance does not hold for children where the sexes are about equally represented. No age is immune to this disease. It has been found in the fetus and in the newborn at postmortem occurring as a congenital condition. It has been diagnosed clinically during infancy. Whether these cases represent congenital bronchiectasis with a superimposed infec-

tion occurring early in life, it is usually not possible to determine without histopathological study of the involved tissues. Approximately half the cases of bronchiectasis start before the age of ten but the disease is usually not recognized until years later. The incidence of bronchiectasis, therefore, increases with each succeeding decade until the third decade when it reaches its peak. Thus bronchiectasis is most common between the ages of twenty to thirty. During the next decade there is a gradual decline in frequency and after forty the disease becomes much less common but is to be found even in the aged. This age distribution distinctly emphasizes the fact that, although the majority of cases of bronchiectasis are diagnosed and treated during adult life, the onset of the condition occurs during childhood. Bronchiectasis is, therefore, primarily a disease of childhood.

PATHOLOGY OF BRONCHIECTASIS

Whenever the bronchial tubes are dilated beyond their normal caliber, there exists the pathological entity known as bronchiectasis. This condition does not ordinarily give rise to symptoms. Only when there is an associated infection do symptoms occur, and not until then can the clinical entity of bronchiectasis be said to exist. An exception to these facts is the occurrence of so-called dry bronchiectasis in which bronchial dilatation but no infection is present. This condition only manifests itself clinically in the occasional occurrence of pulmonary hemorrhages, so that the condition is also known as hemorrhagic bronchiectasis.

That inflammation of one or more bronchi initiates the process of acquired bronchiectasis is fairly well accepted by most authorities. This inflammation is almost invariably the result of infection. Upon the severity and extent of the infection will largely depend the subsequent course of events. If the resulting inflammation is severe enough to cause a desquamation of the epithelial lining of the bronchus with destruction of the underlying muscular and elastic tissues, then the groundwork for bronchiectasis has been laid. This ulcerative process may extend beyond the bronchus into the surrounding parenchyma of the lung. When healing is attempted by the regenerative powers of the lung, granulation tissue, rich in blood vessels, makes its appearance. These granulations are subsequently covered by a somewhat modified type of epithelium, without cilia. The bronchial wall is thus composed of this newly epithelized granulation tissue devoid of muscle and elastic fibers. The granulation tissue is subsequently replaced by fibrous tissue and this may extend for a variable distance into the lung parenchyma. The bronchial wall is thus considerably weakened and dilatation occurs resulting in bronchiectasis.

Bronchiectasis is a progressive disease since each pathological alteration predisposes to additional changes which intensify the condition; thus a vicious circle is created. The changes in the bronchial wall, the loss of the ciliated epithelium and the accompanying fibrosis prevent the elimination of bronchial secretions. The retained secretions become infected and intensify the changes already present. The disorderly and unrestrained production of granulation tissue, which subsequently may result in permanent folds, often causes partial or complete obstruction of the bronchial lumen, thus definitely interfering with respiratory function. The ever present infection in bronchiectasis predisposes to the frequent occurrence of pneumonitis. This complication aggravates the existing bronchiectasis, tends to spread the infection to adjacent portions of the bronchial tree and upper respiratory tract and eventuates in a chronic interstitial or indurative pneumonia as well as a chronic tracheobronchitis.

As a result of the many factors involved, various types of deformity of the bronchi occur. These have been described as cylindrical, fusiform and saccular. The peribronchial and interstitial fibrosis resulting from bronchiectasis tends to distort and in-

crease the bronchial dilatation by direct traction upon the bronchial wall. At times the ulcerative bronchitis is so severe that the bronchial wall is perforated and cavities occur in the surrounding lung parenchyma. These cavities simulate the saccular dilatations of the bronchi. This condition is rightfully referred to as multiple lung abscess or chronic pulmonary suppuration which is often associated with or perhaps the cause or result of bronchiectasis. Finally gangrene of the lung may occur in association with bronchiectasis. The pathological changes as described are for the most part irreversible since the basic changes are primarily the destruction of tissues which show little tendency to regenerate.

The pathological changes of bronchiectasis vary considerably in extent and severity. These changes may be unilateral or bilateral, or they may involve only a small portion of a lobe. It is an interesting observation of Perry and King[20] that bronchiectasis shows little or no tendency to spread beyond the original site of involvement and that the pathological changes are usually maximal when first demonstrated. The fact that patients will become progressively worse clinically is due to the increase in the severity of the accompanying infection and the long continued toxemia which is so often present.

Most statistical reports of the anatomical distribution of bronchiectasis indicate that the left lower lobe is most frequently involved. This is particularly true in children. According to Leopold,[19] bronchiectasis in the left lung is three times as common as in the right in children and twice as common in adults. This predilection for the left lower lobe has been explained by Duken and von den Steinen[8] on the basis that the right lower lobe drains better than the left because the right main bronchus is a direct continuation of the trachea, that the left main bronchus is narrower than the right and shows a constriction where it is crossed by the left pulmonary artery and that in children this artery can readily compress the bronchus. These factors favor lobar collapse and retention of secretions in the left lower lobe.

Pathogenesis of Bronchiectasis

Infected, dilated bronchi constitute the basic pathological changes in bronchiectasis. The manner in which the bronchi become infected is readily understood. The weakening of the bronchial wall as the result of the pathological changes due to infection permits the bronchi to dilate. But what is the force which actually dilates the bronchi? This, without doubt, has been the most controversial aspect of bronchiectasis since the days of Laennec. Many theories have been advanced and although it is believed by some that the problem has been solved, there still continue differences of opinion. The changing views regarding the dilating force in bronchiectasis has been chronologically summarized by Hamman[15] as follows:

> This matter of the dilating force, which leads to bronchiectasis, when the bronchial wall is weakened by disease, has been commented upon and questioned by all authors who have written upon the disease. Medical literature is full of these discussions. Laennec, the first to describe the pathological anatomy of bronchiectasis, ascribed the condition to retained bronchial secretion. Andral, in 1827, emphasized the effects of inflammation upon the bronchial wall leading to attenuation and loss of elasticity. Apparently he thought the stress of ordinary breathing sufficient to dilate such weakened bronchi. Reynard, in 1838, apparently was the first to describe the association of bronchiectasis and bronchial obstruction. Corrigan, 1838, in his studies on cirrhosis of the lung, noted the frequent association of bronchiectasis with extensive fibrosis. He explained the bronchial dilatation as a result of the pull of the scar tissue. Rilliet and Barthez, 1843, were the first to call attention to the frequent occurrence of bronchiectasis in children and to emphasize the role of pneumonia, particularly pneumonia complicating measles and pertussis. Mendelsohn, 1845, contended that cough was the dilating force which produced bronchiectasis when the bronchial walls were weakened by disease. Barth, 1856, stressed again the importance of bronchial obstruction, contending that

all the factors previously invoked to explain the development of bronchiectasis were most effective in the presence of bronchial obstruction. Biermer, 1860, in his comprehensive review of bronchiectasis collected all the views previously maintained and stressed again the importance of bronchial obstruction. During the following sixty years, these opinions have been reviewed and revised with much discussion, and even today the discussion continues. However, during the past fifteen years accumulating evidence has favored the view that bronchiectasis is caused only by bronchial stenosis.

Thus, it is seen that before bronchiectasis can develop, not only is infection necessary but some form of bronchial obstruction must be present. Either bronchial infection or obstruction may be primary. When infection occurs first, bronchial occlusion may result from thick, inspissated secretions, granulation tissue or cicatricial changes in and about the bronchi. When bronchial obstruction is primary, the stagnation and retention of secretions invite infection.

By what mechanism does bronchial obstruction cause bronchial dilatation? When a bronchus becomes completely occluded, collapse of the pulmonary tissue peripheral to the obstruction occurs. The surrounding lung tissue becomes overaerated in order to occupy the space left by the collapsed tissue. Thus, there is created a disturbance in the pressure relationships within the chest, the effect of which is to exert traction upon the infected and weakened bronchi of the collapsed tissue sufficient to cause dilatation. When the lumen of a bronchus is only partially occluded there may be a ball-valve effect; that is, air readily enters the involved tissues during inspiration but is prevented from escaping by the bronchial occlusion during expiration. Thus, with each inspiration there is an ever increasing pressure within the involved lung tissue which may grow to considerable proportions. This is more than sufficient to dilate the weakened bronchi. With partial obstruction, in the absence of a ball-valve arrangement, bronchial dilatation will also occur. Since the inspiratory phase of respiration is more forceful than the expiratory phase, an increased pressure within the tissues supplied by the occluded bronchus, sufficient to dilate the bronchi, will occur. It is believed that in the presence of normal bronchi these forces are not sufficient to cause permanent dilatation. These effects of bronchial dilatation are more effective when the larger bronchi are involved since much more tissue becomes collapsed and the disturbance of intrathoracic pressure is greater. This fact is in conformity with the pathology of bronchiectasis since it usually is the larger and middle sized bronchi that are involved.

ETIOLOGY OF BRONCHIECTASIS

I. *Congenital*
II. *Acquired*
  A. *Bronchial Infection*
    1. *Acute tracheobronchitis*
    2. *Pneumonia*
      a. Bronchopneumonia
        (1) Influenza
        (2) Measles
        (3) Pertussis
      b. Lobar pneumonia
    3. *Chronic paranasal sinusitis*
    4. *Allergy*
  B. *Bronchial Obstruction*
    1. *Intrabronchial*
      a. Foreign body
      b. Broncholith
      c. Occluding secretions
    2. *Bronchial*
      a. Stenosis
        (1) Congenital
        (2) Acquired
          (a) Traumatic
          (b) Infectious
            Specific
            Nonspecific
      b. Tumor
    3. *Peribronchial*
      a. Enlarged lymph nodes
      b. Tumor
      c. Pulmonary fibrosis
  C. *Miscellaneous*
    1. *Lesions of the pleura*
    2. *Lesions of the thoracic cage*

Congenital Bronchiectasis. Although there has been considerable controversy regarding the existence of congenital bronchiectasis, the finding of dilated bronchi in the lungs of the fetus and in newborn infants at autopsy leaves little doubt that this condi-

tion does occur. In the absence of infection it is highly improbable that these cases would give rise to symptoms. When infection is present it is difficult to determine whether the bronchial dilatation is congenital or acquired. The mere fact that clinically demonstrable bronchiectasis may occur a few weeks after birth does not justify the assumption that the condition is congenital since it has been amply shown that under proper conditions bronchiectasis may develop quite rapidly. The etiological factors underlying the production of congenital bronchiectasis are but little understood.

Acquired Bronchiectasis. In acquired bronchiectasis it is generally conceded that bronchial infection and bronchial obstruction are the most important etiological factors. There is the growing belief that a combination of the two is present in every case and that one without the other would probably not result in bronchiectasis. This would account for the fact that the majority of cases of either infection or obstruction do not give rise to bronchiectasis. Either infection or obstruction may be the primary lesion, the other resulting as a complication.

This etiological origin directs attention to the fact that bronchiectasis is never a primary disease but is always secondary to some pre-existing condition. The primary disease may dominate the clinical picture, bronchiectasis occurring as an unimportant complication such as is the case in pulmonary tuberculosis or in most cases of lung abscess. In other instances, however, the primary condition may quickly subside; bronchiectasis appears years later and is then regarded as an independent disease. Such is often the case in bronchiectasis which follows pneumonia.

A. *Bronchial Infection.* From the standpoint of infection an acute tracheobronchitis, either alone or as an accompaniment of a pneumonia, is the basis for the initiation of the changes leading to bronchiectasis. In the history of bronchiectatic patients the onset from a specific illness is to be found in more than half the cases. Warner[24] reported 59 per cent, Diamond and Van Loon[7] 63 per cent. The condition which appears most prominent in such histories is pneumonia (Raia[21] 67 per cent, Warner[24] 30 per cent). Of the different types of pneumonia, bronchopneumonia is the most common offender. In a severe attack there may be an accompanying ulcerative bronchitis. In less severe cases of acute tracheobronchitis and bronchopneumonia, the patient may be left with a chronic tracheobronchitis and develops a predisposition to repeated attacks of respiratory tract infection which eventuate in a bronchiectasis.

Acute tracheobronchitis and bronchopneumonia are frequent complications of such common infections as influenza, measles and whooping cough. As such they may lead directly into bronchiectasis or may leave the patient with a chronic tracheobronchitis. Influenza, in particular, is regarded by some investigators as especially prone to result in bronchiectasis. It is to be remembered that in these acute respiratory tract infections it is not the infection alone which causes the bronchiectasis but the patchy areas of atelectasis due to occluding secretions that supply the mechanism for the dilatation of the bronchi in the involved areas.

Whereas the preceding conditions are common etiological factors to be found in children, lobar pneumonia is occasionally responsible for the bronchiectasis having its onset in adult life. It is common knowledge that atelectasis of varying degree is an essential part of the pathological changes occurring in a lobar consolidation. Many authors, with varying degrees of emphasis, have directed attention to the importance of atelectasis in the production of bronchiectasis. The large bronchi involved in an area of atelectasis do not become collapsed. They thus become subjected to the stress and strain of the respiratory movements of the chest and, weakened by infection, dilatation readily occurs. This is particularly prone to occur in slowly resolving pneumonias.

Lobar pneumonia less commonly results in a chronic tracheobronchitis but in this regard it may also initiate the vicious circle of respiratory tract infections leading to bronchiectasis. Failure of complete resolution in lobar pneumonia is of common occurrence. The local bronchitic and parenchymal changes that constitute the pathological picture of an unresolved pneumonia are such as would readily terminate in a bronchiectasis.

When one thinks of a lung abscess, instinctively the possibility of a tonsillectomy as the etiological factor is considered. This association is a well established medical fact. That bronchiectasis may occur as a complication of tonsillectomy is less generally known. It is, however, an etiological factor which must be given some consideration. In the 400 cases of Perry and King,[20] 43, or 11 per cent, followed tonsillectomy and adenectomy. This type of bronchiectasis probably also originates as an acute respiratory tract infection, either in the form of a bronchopneumonia or lobar pneumonia.

Chronic Paranasal Sinusitis. Considerable attention has been given to the relationship of disease of the paranasal sinuses and pathological conditions of the lung. That such a relationship exists there can be no doubt but as regards bronchiectasis some of the more recent writers have taken issue with those authorities who maintain that sinusitis is one, if not the most important, predisposing cause of bronchiectasis. Goodale[12] and also Clerf[6] have emphasized the importance of sinusitis in the etiology of bronchiectasis. Erb,[9] on the other hand, is of the opinion that sinusitis follows rather than precedes bronchiectasis in most instances. Nevertheless it cannot be denied that sinusitis is an important factor in the development of bronchiectasis. The frequency of the "common head cold" with involvement of the paranasal sinuses, the tendency of the sinuses to harbor infection and to become chronically involved and the tendency of the chronically infected sinuses to act as a source of reinfection for the rest of the respiratory tract leading to a chronic tracheobronchitis has been well established. This predisposes to attacks of pneumonia from which bronchiectasis may develop. The fact that many individuals with chronic sinusitis never develop clinical evidence of bronchiectasis is no argument against the relation of the two conditions.

The frequent association of sinusitis and bronchiectasis has been an outstanding feature of many statistical studies. Thus, Farrell[10] found 86 per cent of his series of bronchiectatic patients with sinus involvement; Diamond and Van Loon[7] report 64 per cent, and Raia[21] 82 per cent. It must be remembered, however, that the mere association of sinusitis with bronchiectasis is no justification for the conclusion that the sinusitis caused the bronchiectasis. There may be no connection whatever. On the other hand, there is no denying the fact that paranasal sinusitis often results from a bronchiectasis. The continual drainage of infected material from the dilated bronchi aggravates the existing bronchitis which spreads by continuity until the entire respiratory tract becomes involved including the nasal accessory sinuses. This sequence of events seems inevitable. The coexistence of sinusitis and bronchiectasis leads to a vicious circle, an exacerbation of the infection in one area resulting in an acute flare-up in the other. This has been convincingly demonstrated by Graham[13] in the study of patients with bronchopleural fistulae wherein it was found that an acute sinusitis resulted in aggravation of the inflammatory process and increase in the secretions from the bronchopleural fistula.

Allergy. One rather hesitates to discuss the relationship of bronchiectasis and allergy of the respiratory tract as manifested in asthma and hay fever. There are those who deny any relationship, while others regard allergy as an important etiological factor in the development of bronchiectasis. Watson and Kibler[25] studied a series of 46 cases of bronchiectasis and found that a large percentage had evidence of allergy. They noted that in the majority of pa-

tients, bronchiectasis was favorably influenced by treatment of the allergy. They directed attention to the close roentgenological resemblance of bronchiectasis and basal allergic bronchitis which can only be differentiated by bronchographic studies. They also stated that the common association of sinusitis and bronchiectasis can be explained on the basis of allergy.

When one considers the effects of hay fever and asthma upon the respiratory tract it would seem logical to expect bronchiectasis to follow as a complication of these conditions. The vasomotor phenomena which occur in the nose and paranasal sinuses as the result of allergy predispose these structures to infection. The common association of chronic sinusitis and hay fever bears witness to this fact. Similar factors operate in the lungs in patients with asthma. In well established cases, a chronic tracheobronchitis is an almost invariable accompaniment. This is readily demonstrated upon roentgen examination of the chest in individuals with asthma. The appearance of a severe chronic tracheobronchitis as manifested by an intensification of the hilar and truncal shadows of the lungs is often sufficiently characteristic to suggest that the patient is asthmatic. That the chronic tracheobronchitis, associated with atelectasis which so commonly occurs in asthma, should not result in bronchiectasis does not seem reasonable since all the factors necessary for its production are present.

B. *Bronchial Obstruction.* There is ample evidence both clinically and pathologically that bronchial obstruction is a prime factor in the etiology of bronchiectasis. The causes of bronchial obstruction are legion and are best considered from the standpoint of their relationship to the bronchus. The mechanism of the production of bronchiectasis by bronchial obstruction has already been considered. We need here merely to give attention to the specific forms of bronchial occlusion.

1. *Intrabronchial Obstruction.* In this category foreign bodies stand out as being particularly significant. All manner of foreign bodies gain access to the lungs and come to rest in one of the larger bronchi where a variable degree of obstruction is produced. With complete obstruction atelectasis occurs distal to the obstruction. With partial occlusion there is retention of secretions producing the so-called "drowned lung" and with a ball-valve type of obstruction there occurs an obstructive emphysema. Atelectasis, retained secretions and emphysema are recognized factors in the etiology of bronchiectasis. Whether or not a foreign body will produce a bronchiectasis s dependent upon several associated factors. Infection accompanying or resulting from the lodgment of a foreign body is necessary and the duration of the sojourn of the foreign body in the bronchus is also of prime importance. The longer the bronchial obstruction, the more severe and extensive the resulting changes and the more likely the development of bronchiectasis. Obviously bronchiectasis is more common in those instances in which there is no definite history of the aspiration of a foreign body, which may have occurred months or years before. This type of case is surprisingly common as revealed by roentgenologic and bronchoscopic studies.

Various writers have directed attention to the significance of broncholiths in the etiology of bronchiectasis. The frequency of this occurrence would be difficult to determine. The mechanism involved is similar to that operative in the case of extraneous foreign bodies.

Bronchial occlusion by mucous plugs and inspissated secretions is of fairly common occurrence and therefore important in the etiology of bronchiectasis. This is the cause of atelectasis in pneumonia and in postoperative patients. Such an occurrence is not uncommon in bronchial asthma nor in bronchiectasis itself. This type of bronchial obstruction may be encountered in almost any type of pulmonary infection.

2. *Bronchial Obstruction.* Lesions which involve the bronchial wall directly are not uncommon. Stenosis of a bronchus may

arise from a variety of causes. It may be congenital and as such may be one of the causes of congenital bronchiectasis. In the acquired variety stricture may be of traumatic origin, such as occurs from the lodgment of a foreign body, particularly those having sharp points. During the removal of a foreign body trauma of sufficient severity may occur to a bronchus to result in a stricture.

Stenosis may also arise during an acute respiratory tract infection from a fairly localized ulcerative bronchitis. With subsequent healing and scar tissue contraction strictures of varying degree may result. The infection may be of specific origin, such as tuberculosis or syphilis. Tuberculous granulations within the bronchi may produce multiple strictures. Syphilis is sometimes referred to in the etiology of bronchiectasis. This is not due to the specific infection itself but to a bronchial stenosis which is prone to occur in this condition.

Tumors of the bronchial wall are being encountered with increasing frequency. They may be either benign, in the form of an adenoma or papilloma or malignant, as a bronchogenic carcinoma. The latter are much more common. These tumefactions readily occlude the lumen of the bronchus, produce atelectasis, invite infection and thus initiate the changes essential for the development of bronchiectasis.

3. *Peribronchial Obstruction.* Peribronchial obstruction occurs as the result of extrinsic pressure upon a bronchus. Anything capable of producing this type of pressure may cause bronchiectasis. While there are many possibilities in this regard only a few of the more common need be mentioned here.

Enlarged lymph nodes occurring in the hilar region, from whatever cause, may produce sufficient pressure upon one or more of the larger bronchi to cause partial occlusion. This interference with pulmonary ventilation favors the retention of bronchial secretions, invites infection and may ultimately lead to bronchiectasis. This sequence of events has been growing in importance with many recent writers on the subject. It affords an adequate explanation for the predominance of the onset of bronchiectasis in children. Enlargement of hilar lymph nodes is of common occurrence in children from such conditions as pertussis and tuberculosis.

Tumors in the mediastinum, hilum or parenchyma of the lung will, by direct pressure or invasion, occlude one or more bronchi and as in the case of enlarged lymph nodes the stage is set for the development of a bronchiectasis. Such tumors may be of various kinds, benign or malignant and include aneurysms of the aorta and the tumefactions of lymphoblastomata.

Pulmonary fibrosis, which is such a common pathological entity, is capable of causing peribronchial obstruction. Corrigan, 1838, regarded cirrhosis of the lung due to pulmonary fibrosis as the cause of bronchiectasis. This theory is not now generally accepted although there remain some adherents to this idea. It has been pointed out that pulmonary fibrosis usually follows rather than precedes bronchiectasis. It is generally agreed, however, that pulmonary fibrosis is a contributory factor in bronchiectasis operating in two ways: by producing peribronchial occlusion and direct traction on the bronchial wall. Outstanding in this respect is the fibrosis so commonly associated with tuberculosis in which bronchiectasis to some degree is an invariable accompaniment. While certain authors have emphasized the pulmonary fibrosis of pneumoconiosis, it is certain that bronchiectasis does not often assume clinical significance in this condition. Possibly the element of infection is not sufficient to produce symptoms of bronchiectasis

C. *Miscellaneous.* Of minor if not of questionable significance are certain miscellaneous conditions to which bronchiectasis is sometimes attributed. Lesions of the pleura, particularly adhesions, are sometimes mentioned as playing an important rôle. Such adhesions are supposed to exert sufficient traction upon the bronchi during

respiratory movements to cause dilatation. It is doubtful whether such a mechanism alone could produce bronchiectasis. Much more probable is the supposition that the pulmonary condition responsible for the pleural adhesions is the causative factor of a resulting bronchiectasis. Significant is the observation that bronchial dilatation does not occur in long continued pneumothorax and apparently bronchiectasis does not occur in long standing pleural effusions in which the lung is continuously compressed.

In much the same category is the relation of congenital and acquired deformities of the thoracic cage to bronchiectasis. Supposedly there is a weakening of the respiratory effort in the presence of thoracic deformities which leads to bronchial dilatation. The bronchiectasis which is sometimes seen associated with such conditions is most probably not due to the thoracic deformity *per se*. Deformities of the chest are at times of such a nature as to interfere with the normal physiology of respiration. This will, of course, predispose to respiratory tract infections and such infections are more probably the cause of any resulting bronchiectasis. Under such conditions, however, the thoracic deformity must be regarded as a contributory factor.

BACTERIOLOGY OF BRONCHIECTASIS

The etiology of bronchiectasis has been repeatedly sought for in the bacteriology of this disease. Claims have been made from time to time that certain organisms are directly responsible for the occurrence of bronchial dilatation. Thus Smith[23] has emphasized the importance of fusospirochetal infection. It is true that the Vincent's organism has a greater destructive effect upon the bronchial wall than most other organisms, but most observers are of the opinion that these organisms are secondary invaders, especially in view of the fact that they are more commonly found in the bronchiectasis of adults than of children. The frequency of Vincent's infection in the mouth of adults is common knowledge. In bronchiectasis a large variety of bacteria are to be found which vary from time to time and seldom is one organism present in pure culture. Most observers agree with Greey[14] that there is nothing specific in the bacteriology of bronchiectasis.

SYMPTOMATOLOGY OF BRONCHIECTASIS

The cardinal clinical manifestations of bronchiectasis are a chronic productive cough, the expectoration of variable amounts of sputum which may or may not be fetid, and occasional hemoptysis. Many inconstant and variable signs and symptoms may be present, such as recurring bouts of fever, dyspnea, night sweats, anemia, loss of strength and weight. The symptomatology is largely dependent upon the extent of the disease and severity of the infection. In the milder forms there may be no symptoms whatever. This is particularly true of so-called dry bronchiectasis in which there is dilatation without infection.

In the typical case of bronchiectasis, because of the constant infection present there is an excess amount of bronchial secretion. In an attempt to eliminate this material a chronic cough develops. This is almost invariably much worse upon arising in the morning due to the accumulation of secretions during the night. Patients often learn that by assuming certain postures drainage of the accumulated secretions can be facilitated. After such drainage the patient may be fairly free of cough for the remainder of the day. The amount of the sputum is a variable one depending upon the severity of the infection. At times it is very copious, thick, tenacious and difficult to raise.

The retained secretions serve as an excellent medium for a host of bacteria, many of them saprophytic in nature. According to Jackson and Jackson,[16] these serve to decrease the viscosity of the sputum so that it can be more readily expelled. This putrefactive process causes the sputum to be very foul and offensive. The odor is said to be due to the presence of a fusospirochetal infection. Considerable emphasis has been

placed upon this feature of the condition since upon this one factor alone the diagnosis of bronchiectasis can be suspected although offensive sputum also occurs in pulmonary abscess, chronic pulmonary suppuration and gangrene of the lung but usually in a more exaggerated form. Although a copious and fetid sputum is characteristic and almost invariably present in the advanced stages of the disease it should be remembered that such sputum is not present in the majority of cases of bronchiectasis. Warner[24] found that in only 20 per cent of his cases was there a daily production of 4 ounces or more of foul smelling sputum. One characteristic which he regards as more important and extremely suggestive of bronchiectasis is the "chunky" nature of the sputum. Nevertheless, the importance of the foul and fetid sputum of bronchiectasis cannot be minimized since it is the one feature of the disease which makes the condition such a loathsome one for both the patient and those with whom he comes in contact. Jackson and Jackson[16] have likened the condition to a septic tank.

It is not sufficiently appreciated that bronchiectasis is one of the most common causes of hemoptysis. This is particularly true in children. Blood spitting occurred in 45 per cent of the cases reported by Warner.[24] To the average physician, bleeding from the lung means tuberculosis. No one has a better opportunity to appreciate this fact than the roentgenologist. A request for a chest roentgenogram in a patient with blood spitting is usually accompanied by a diagnosis of tuberculosis. The two conditions, aside from tuberculosis, which must be looked for in the roentgenogram in such cases are bronchiectasis and mitral stenosis. It is surprising the number of such cases encountered by the roentgenologist in which the diagnosis of tuberculosis has been made by the clinician.

If one recalls the pathology of bronchiectasis the basis for hemoptysis is readily understood. The fresh, highly vascularized granulation tissue which is so abundantly present in bronchiectasis shows a tendency to bleed quite readily. The irritating, harassing cough produces sufficient trauma to these granulations to cause them to bleed occasionally. The bleeding is usually not very great in amount. A mere streaking of the sputum with blood may be all that is present but it may recur quite frequently. At times, however, quite a profuse hemorrhage may occur and this may prove to be a serious complication.

Depending upon the amount of bronchial secretions and the difficulty with which it is eliminated, there may be a low grade fever and the patient may become septic. With the establishment of adequate drainage these symptoms disappear. The constant absorption of septic material produces a state of low grade toxemia. This eventually leads to anorexia, a poor state of nutrition, a progressive anemia, and loss of weight and strength. Since bronchiectasis is a progressive disease there is an inevitable impairment of respiratory function leading to a variable degree of dyspnea. The inevitable myocardial damage resulting from the increased resistance within the lesser circulation incident to the pulmonary fibrosis of bronchiectasis plus the prolonged state of toxemia adds materially to the patient's signs and symptoms.

PHYSICAL EXAMINATION IN BRONCHIECTASIS

In comparison with the extent and severity of the pathological changes the physical signs in bronchiectasis are rather negligible. There are no signs which are pathognomonic. In the more advanced cases some restriction in the movement of the chest may be noted, there may be some dullness to percussion over the involved portions of the lung and the breath sounds may be somewhat less audible. Râles occasionally may be heard but are by no means constant. In long standing cases clubbing of the fingers characteristic of chronic pulmonary suppuration appears and the changes of chronic pulmonary osteoarthrop-

athy may be present. Eventually amyloidosis may develop.

DIAGNOSIS OF BRONCHIECTASIS

Only in the advanced cases are the symptoms sufficiently characteristic to warrant a diagnosis of bronchiectasis. In the mild and moderately advanced cases, which constitute the vast majority of patients, the symptoms may be merely suggestive. Of greater significance is the history of a definite onset during childhood from some specific infection, leaving the patient with a predisposition to frequent respiratory tract infections, a chronic cough which gradually becomes worse, accompanied by an increasing amount of expectoration. With such a history the diagnosis of bronchiectasis is most probable and a roentgen examination of the chest is in order.

The ordinary roentgenogram of the chest will not, as a rule, warrant a positive diagnosis of bronchiectasis. When this disease can be recognized in the ordinary roentgen study, the condition has reached an advanced stage. When viewing roentgenograms in a suspected case of bronchiectasis the possibility of a foreign body should never be forgotten. Quite characteristic of bronchiectasis are the dense triangular shadows frequently found in the base of a lung adjacent to the heart. Usually an intensification of the hilar and trunk shadows especially those to the bases is all that is found in the plain roentgenogram. This is indistinguishable from a chronic tracheobronchitis. Such findings in a child or young adult combined with a history as aforementioned warrants a suggestive diagnosis of bronchiectasis. The next step in the investigation is a bronchoscopic examination.

A bronchoscopic examination is an absolute essential in every patient with suspected bronchiectasis. This will reveal information not obtainable in any other way. One sees the condition of the respiratory tract from the larynx to the larger bronchi and learns whether these parts are involved by the inflammatory process and the extent and severity of this involvement. The presence of congenital anomalies can be detected, and important in every case of bronchiectasis is the presence of a foreign body. The actual site of the bronchiectasis is not reached by the bronchoscope so that the bronchial dilatations are rarely seen. The character of the inflammatory reaction and the aspiration of considerable amounts of thick, viscid, purulent secretions from the smaller bronchi are highly suggestive of bronchiectasis. Thus, while a definite diagnosis of bronchiectasis cannot be made by bronchoscopic examination, except in the advanced cases, the findings are usually such as to indicate the need for bronchography

The roentgen visualization of the bronchial tree after the introduction of a contrast medium is becoming of increasing value in all manner of pulmonary conditions. In bronchiectasis it is the only absolute method of establishing the diagnosis regardless of the stage of the disease. A number of methods have been devised for introducing the iodized oil into the bronchial tree. All give good results depending upon the skill of the physician performing the procedure. By means of bronchography the extent and nature of the bronchial dilatation are readily ascertained. It can be learned whether the disease is sharply localized, whether it involves more than one lobe and whether it is unilateral or bilateral.

In the final analysis, therefore, while there are many factors which may lead the clinician to suspect bronchiectasis, the diagnosis must actually be established by bronchography.

CLINICAL COURSE AND PROGNOSIS OF BRONCHIECTASIS

Bronchiectasis is primarily a disease of childhood, runs a slow progressive course, becomes clinically established during adolescence or early adult life, leads to a life of chronic invalidism and terminates in death from one of a number of complications. The condition has its onset during

childhood resulting from a combination of respiratory infection and bronchial occlusion. The disease may run a very rapid course becoming well established and easily recognized within a few weeks or months. The usual course of events, however, is much more insidious, the patient enjoying comparatively good health for many years after the onset of the condition but showing a greater predisposition to respiratory tract infections than the average individual. These infections increase in frequency and severity until adult life is reached when a well established bronchiectasis has resulted. In about two-fifths of such cases the initial condition responsible for the bronchiectasis has long since been forgotten.

The subsequent history regardless of whether the development has been rapid or slow is fairly characteristic. The patient's general health may be good but there is a tendency to "take cold" rather easily. These "colds" become increasingly difficult to "throw off." The patient is left with a persistent, annoying cough in between attacks of respiratory tract infection. The cough becomes somewhat productive. The sputum increases in amount and eventually becomes fetid and foul in character. The patient has increasing difficulty in raising the sputum and soon learns that this can be facilitated by assuming certain positions. His symptoms are worse in the morning. He never feels quite up to par and at times he is distinctly toxic. His general health begins to fail, he suffers from lassitude and ready fatigability, there is a loss of appetite and gradual loss of weight and strength. The development of this state of affairs may occur rapidly in the course of a few years or, on the other hand, the progression may be very slow covering a period of twenty to twenty-five years.

Eventually other systems of the body become involved, especially the cardiovascular system, the symptoms of which are superimposed upon those of the bronchiectasis. The terminal event is usually in the form of some complication which is fairly common. The patient becomes subject to repeated attacks of pneumonia. In Warner's[24] series 52 per cent had at least one attack of pneumonia and 27 per cent had more than one attack. One such attack may be severe enough to end fatally. Cardiac failure is a frequent cause of death in long standing cases. Massive pulmonary hemorrhage is the cause of a fatal termination in some cases. Brain abscess is a notorious complication in suppurative conditions of the lung and usually proves fatal. General debility and asthenia with a terminal hypostatic pneumonia may be responsible for the bronchiectatic demise.

Bronchiectasis is, however, not necessarily fatal. Although it is a progressive and irreversible disease it may at times become arrested or its progress may be so slow that the patient may live his allotted span of life and die from some other cause. In general, however, the prognosis of untreated bronchiectasis is poor.

The prognosis of bronchiectasis varies considerably in individual cases. Warner, in a series of 110 cases, reported a mortality of 23 per cent, with the average duration of the disease of nine years. Roles and Todd[22] report a general mortality, regardless of treatment, of 38 per cent. The mortality rate as reported by Perry and King[20] was 31 per cent. In the series of 171 cases reported by Bradshaw, Putney and Clerf,[5] 34.5 per cent died from bronchiectasis or its complications, with the average duration of the disease thirteen and a half years.

Economic Importance of Bronchiectasis

As has been stated, the patient with a definitely established bronchiectasis becomes a chronic invalid. The frequently recurring attacks of respiratory tract infection cause much loss of time from work. This may make it difficult for an individual to hold a worth while position. The harassing cough and particularly the offensive sputum makes the individual objectionable to those with whom he is required to work. The toxicity and general lassitude so commonly present definitely interfere with the

individual's efficiency. He becomes painfully aware of his condition, of his inability to maintain his place in the world and to meet his social and economic obligations. This state of affairs has in some instances been terminated by suicide. It is important that physicians should recognize these facts. Thus the need for the prevention of bronchiectasis becomes all the more apparent.

TREATMENT OF BRONCHIECTASIS

Until recent years, the treatment of bronchiectasis was entirely medical. The most important single item in this form of therapy is postural drainage to rid the lungs of their septic material and to prevent toxemia. Many patients can be kept fairly comfortable by this one method alone. Sedation to control the annoying cough and to insure the patient rest and sleep is an important factor in treatment. Expectorants may aid in expelling the thick, tenacious sputum. In the presence of fusospirochetal infection the intravenous injections of neoarsphenamine have been found useful, especially in overcoming the fetor of the breath. Creosote by mouth has been used for the same purpose. General supportive measures to maintain health is of vital importance and must be combined with whatever therapy is employed. Precautionary measures to prevent or minimize the severity of the recurrent respiratory tract infections are of the utmost importance. As will be recognized, medical treatment is but palliative and not curative. No specific therapy in the form of sera or vaccines has been found of any avail. There has not yet been sufficient experience with the use of sulfonilamide and its derivatives to know whether or not these drugs will be of benefit in the treatment of bronchiectasis.

With the advent of the bronchoscope a new form of therapy for bronchiectasis was introduced, namely bronchoscopic drainage. This has been of inestimable value. It accomplishes in a much more efficient manner what the physcian has attempted to do with postural drainage. There is no question that the patient relieved of the germ laden secretions will be very much improved, subjectively and objectively, the progress of the disease will be much delayed and some of the complications may be prevented. The direct injection of drugs into the bronchial tree has not as yet proved of definite value, although there is the impression that patients are sometimes improved after the use of lipiodol for bronchography. Bronchoscopic drainage does not materially alter the underlying pathological changes of bronchiectasis; therefore, the condition is not cured by this form of therapy.

In 1934 Berck[1] reported his experience with radiation therapy in the treatment of bronchiectasis. This was followed by a second paper in 1937 by Berck and Harris[2] in which they reported definite improvement in a large percentage of the patients treated. According to these authors, the rationale for the use of this method is an attempt to change an infected and overproductive type of bronchiectasis into the dry form of the disease. It is well known that if the secretions can be minimized there is a marked improvement in the patient's symptoms and general physical condition. This method of therapy has not become very popular and there are few reports to substantiate the claims of the originators of the method.

The most recent addition to the therapy of bronchiectasis is surgery in which the involved lobes of the lung are removed. Other forms of surgery such as pneumothorax, phrenicectomy and thorocoplasty have also been tried but none of these have been of permanent benefit except in isolated cases.

The success of lobectomy and pneumonectomy is dependent upon a number of factors, such as the proper selection of cases, the general condition of the patient and the skill of the surgeon. The most favorable cases are those in which the disease is unilateral and limited to one lobe. The least favorable are those in which the disease shows a widespread bilateral involvement.

In these, even though the lobes most involved are removed, the entire respiratory tract is more or less involved by the underlying inflammatory process responsible for the bronchiectasis. In such cases, although considerable palliation can be accomplished by surgery, actual cure of the condition is impossible.

The general condition of the patient is of the utmost importance in the surgical treatment of bronchiectasis. Obviously a patient who is much debilitated, emaciated and anemic is a poor surgical risk and will not have the stamina nor the recuperative powers to withstand a formidable operation. Therefore, it is important that operation be not too long delayed and should be done before the patient begins to show systemic effects of the disease. In some advanced cases it may be necessary to restore the patient to an improved physical state before operation is attempted. Certain it is that surgical results will be much better the earlier operation is performed; this means a resort to surgery during childhood in many if not most of the cases. It is difficult to convince the referring physician to submit his patient to a major thoracic operation for a disease which in its earlier stages does not appear to be so very serious. Much education of the general medical profession regarding bronchiectasis is needed before the general practitioner will understand and have the proper attitude toward the surgical treatment of this disease.

The question arises as to whether surgery should not be done as soon as the diagnosis of bronchiectasis has been established. In view of the variable course of the disease in different individuals it would appear more logical to keep the patient under observation for a time and attempt to control the progress of the condition by less radical means. While definite progression is the rule in clinical bronchiectasis, at times this progression is so slow that the condition never becomes of serious import. Conservative measures should not be persisted in if the patient does not show definite improvement or the disease does not become arrested within a reasonable period of time.

Finally, one must consider the fact that a lobectomy or pneumonectomy carries with it a certain mortality. According to Bradshaw, Putney and Clerf,[5] the general mortality is about 10 per cent. In unilateral involvement the mortality is under 5 per cent. This is a matter which must be seriously considered when operation is advised. Furthermore, complications, such as empyema and bronchopleural fistula, may occur. Thus the skill of the surgeon plays a vital part in the success of the surgical treatment of bronchiectasis. The thorax is one of the most recent parts of the body to be invaded by the surgeon. Not all surgeons are trained in chest technique; therefore mortality statistics will vary widely. It must be remembered that only the expertly trained thoracic surgeon publishes his mortality statistics. Thus, the choice of a surgeon becomes vitally important when operation for bronchiectasis is being considered.

Surgery has outmoded the statement of Boyd,[3] "Once bronchiectasis is well established in childhood an early death from bronchopneumonia can only be regarded as a blessing." While surgery has very definitely improved the prognosis of bronchiectasis, surgery does not solve the problem of this disease since it is admitted by all that there are many cases not suitable for surgery. No precise statement has been found indicating the percentage of patients with bronchiectasis suitable for surgical treatment. Inquiry of several thoracic surgeons reveals a wide difference of opinion, ranging from 25 to 75 per cent. Bradshaw,[4] from his large experience in the surgical treatment of bronchiectasis, believes that not more than 25 per cent of patients are suitable for surgery when the disease is first diagnosed. He is impressed with the advanced stage of the disease when the patient first seeks medical attention, even when the history of the condition is of relatively short duration.

If one were arbitrarily to assume that

only unilateral cases of bronchiectasis are suitable for surgery, which is of course not true, some idea as to the percentage of patients in whom surgical treatment could be used might be obtained by reference to the anatomical distribution of the disease as reported by various authors. Thus, of the 979 cases collected from the literature by Leopold,[13] in 686 the disease was unilateral. Therefore, approximately 70 per cent would be suitable for surgery. There is considerable range among the authors quoted; thus, in the 60 cases of Block and Francis, 53 per cent were unilateral while in the 167 cases reported by Duken and von den Steinen, 81 per cent were unilateral. This latter series included children only. From this method of deduction it would appear that for about 30 per cent of patients with bronchiectasis there is at present no hope of cure. This further emphasizes the necessity of prevention.

This discussion of treatment has been based upon the generally accepted concept that bronchiectasis is a progressive and irreversible disease. No consideration has been given to the condition referred to by a number of writers under the name of pseudobronchiectasis (Jackson) and reversible bronchiectasis (Lander and Davidson,[18] Fleischner[11]). If it can be established that the condition mentioned is definitely clinical bronchiectasis then our entire concept of the disease must be revised. It would no longer be proper to refer to bronchiectasis as irreversible; spontaneous, medical, bronchoscopic and even radiation cures could be expected and the postponement of surgical treatment would appear more justifiable. In the present state of our knowledge of this condition the term "pseudobronchiectasis" would appear quite appropriate.

With this consideration of treatment, we have before us the full life story of bronchiectasis. With this knowledge of the disease, its importance can be more readily appreciated. There is little wonder that many have come to regard bronchiectasis as one of the really important diseases of the respiratory tract. It is rapidly assuming an importance equal to that of tuberculosis. While the latter disease is now fairly well understood, its methods of propagation known, its detection and recognition relatively simple and, what is more important, and methods of treatment now so perfected that there is a steady decline in the mortality rate, it can be hoped that this disease soon will be definitely under control. This is not true of bronchiectasis, however. There is yet much to be learned, and until our knowledge of bronchiectasis is more complete, not too much can be hoped for from our present methods of combatting it. It is for this reason that the prevention of bronchiectasis is of such vital importance.

PREVENTION OF BRONCHIECTASIS

Measures for the prevention of bronchiectasis must be based upon a knowledge of its etiology. Thus in discussing prevention a review of the etiological factors concerned becomes essential.

Obviously nothing can be done to prevent the cases of congenital bronchiectasis. However, since these cases are so few in number their clinical significance is relatively negligible.

In the acquired form of bronchiectasis, bronchial infection stands first and foremost among the causes of this condition. This is particularly true in children. Here the laity, particularly mothers, must be made conscious of the seriousness of the common cold and must be educated in the modern methods of cold prevention as far as this is possible. There must be maintenance of child health at the peak of perfection by proper nutrition, with inclusion of all essential food elements and with special consideration of the various vitamins. There must be insistence upon the proper amount of food, rest and sleep, the avoidance of overfatigue, adequate fresh air and sunshine, proper protection against inclement weather and the avoidance of contact with those suffering from acute respiratory tract infec-

tions. Children with colds should be kept away from school. The prompt attention and proper treatment of the common cold by parent and physician will minimize the likelihood of the development of bronchopneumonia. Such education is the responsibility of every practitioner called upon to treat children. The utilization of women's clubs and parent-teachers associations can serve as vehicles for this educational program.

Since the onset of bronchiectasis is so frequently attributed to an attack of influenza, measles or whooping cough, the physician should take every precaution to prevent or to minimize bronchopneumonia occurring as a complication. A more prolonged or more guarded convalescence in these diseases may aid materially in such prevention. Parents too often fail to appreciate the significance of these diseases of childhood and do not heed the warning of the physician concerning complications. Strict adherence to the treatment prescribed must be insisted upon by the attending physician.

While lobar pneumonia may not be one of the most common causes of bronchiectasis, this possibility occurs with sufficient frequency to demand the attention of the clinician. Incomplete resolution of the pneumonic consolidation is far more common than the average physician appreciates. The roentgenologist has ample opportunity to be familiar with this fact. In those institutions in which cases of pneumonia are closely followed by serial roentgenograms of the chest, the roentgenologist has become impressed with the manner in which the roentgen signs of pneumonia lag behind the physical signs. Clinically, the patient may have made a complete recovery, is without physical signs or symptoms and yet on the roentgenogram there remains evidence that resolution is not yet complete. It is usually at this stage that the patient is discharged from the hospital. Presumably these residual roentgen manifestations disappear in the vast majority of cases. Undoubtedly, however, many of these patients return years later with a well established bronchiectasis at the site of the former pneumonia. This accounts for many of the unilateral cases of bronchiectasis. The prophylactic measure here indicated is quite obvious. In all cases of pneumonia of any variety, if the lungs are not clear when the patient is discharged from the hospital he should be required to return for subsequent roentgen studies. If the abnormal signs persist, a bronchoscopic examination should be made and treatment instituted as indicated. In this manner many cases of bronchiectasis could be prevented.

It is realized that the majority of patients with pneumonia are treated at home without the benefit of roentgen studies. As soon as such patients become ambulatory they should be referred for a roentgen examination of the chest. It is surprising the amount of pulmonary disease that would be revealed if such a procedure were adopted as a routine in every case of pneumonia. Not only would a certain amount of bronchiectasis be prevented but other unsuspected and serious pulmonary lesions would come to light such as tuberculosis and bronchogenic cancer which were directly responsible for or erroneously diagnosed as pneumonia. Also early cases of bronchiectasis itself would be detected in time to prevent the serious consequences of this disease.

The argument is valid that many cases of lobar pneumonia in adults are the result rather than the cause of bronchiectasis. This point is often missed by the clinician in treating pneumonia. If roentgen studies were continued in all cases of pneumonia until the lungs were clear, these unsuspected cases of bronchiectasis responsible for attacks of pneumonia would be revealed and immediate treatment could be started rather than postponing such therapy until the diagnosis of bronchiectasis is all too obvious. When a patient with pneumonia gives a history of having had three or four previous attacks, one can be certain that there is some local cause for

this recurrence. All such cases should certainly be followed after recovery from an attack of pneumonia. Many such patients will be found to harbor an unrecognized bronchiectasis.

Regardless of one's opinion as to the relationship of infections of the paranasal sinuses and bronchiectasis, certainly the patient should be given the benefit of the doubt and the disease in the sinuses eradicated if possible. It should be constantly borne in mind that in every acute respiratory tract infection, every part of this tract is involved even though the symptoms are limited specifically to the lungs, trachea or pharynx. It should be remembered that the sinuses are almost certainly involved and some attention should be given to them. This is advice fraught with some danger since the sinuses will not tolerate too enthusiastic and unskilled treatment. Often they are best let alone, since normal sinuses having adequate drainage during an acute respiratory tract infection will usually take care of themselves. However, the sinuses should not be neglected and when obviously involved should receive appropriate treatment. The practitioner who is not too sure of himself about the paranasal sinuses should consult a specialist.

In a chronic sinusitis in which complete eradication of the disease is practically impossible, measures should be taken to minimize the significance of the pathological changes and to prevent acute exacerbations. This is usually a problem for the rhinologist. And particularly is it a problem in the so-called cases of bronchosinusitis which are the ones most likely to develop bronchiectasis. Often little can be done for these patients without a change in climate. They usually do better and often live in comfort in a warm, dry, equable climate such as is to be found in the southwestern part of this country. The problem is not merely a local one and the general health of the patient must be carefully considered.

The fact that a definite relationship between allergy and bronchiectasis has not been fully accepted should not deter the physician from thinking of allergy in his connection. In patients having frequent respiratory tract infections, especially in those in whom the usual measures for combatting such a predisposition have failed, allergy as the underlying cause should be considered. There are those who believe that the predisposition to frequent "colds" is always an allergic manifestation. Thus a consciousness of allergy and active treatment of this phenomenon when found can, in the present state of our knowledge, be regarded as measures in the prevention of bronchiectasis. In frank cases of hay fever and asthma, the measures taken to combat these conditions automatically act prophylactically against bronchiectasis.

Bronchial obstruction is the other important factor to be considered in acquired bronchiectasis. Such obstruction may arise from something within the lumen of the bronchus. Foreign bodies either radiopaque or non-opaque, are common examples of this kind. All too often the inhalation of a foreign body is too lightly regarded by both the patient and his physician. The absence of symptoms leads to the conclusion that the foreign body has entered the alimentary tract and that nothing, therefore, need be done about it. After an initial period of gagging and coughing, a patient with a foreign body in a bronchus or in the pulmonary tissues may become symptomless. If neglected it is certain to give rise to infection and usually terminates in a localized bronchiectasis. In every instance of foreign body a careful roentgen examination of the neck, chest and abdomen is indicated. Because a foreign body is known to be non-opaque, many physicians believe that a roentgen study is useless. Non-opaque foreign bodies, as a rule, are quite readily detected in the respiratory tract and in the esophagus. Too many physicians are inclined to belittle the importance of the swallowing of a foreign body and often roentgen studies are done upon the insistence of the patient. The roentgen examination should be carried out as soon after the inhalation of the foreign body as is

practical. The damage done by a foreign body in the lung is, in a measure, dependent upon the duration of its sojourn and should therefore be removed at the earliest possible moment.

The problem of foreign bodies in the respiratory tract is particularly important in the case of children. In such patients, a history is often lacking, due to the fact that in infants no one may have seen the child swallow a foreign body and in the case of older children they are often afraid to tell of such an incident. Such cases are fairly common in roentgenological practice, and unfortunately many such patients do not reach the roentgenologist for months or years after the aspiration of a foreign object. By this time a bronchiectasis has usually become well established. In a child having repeated respiratory tract infections or in one already showing symptoms suspicious of bronchiectasis the possibility of a foreign body should be considered. In such cases a careful roentgen study should be carried out and in the event of a negative report a bronchoscopic examination should be done to absolutely rule out the presence of a foreign body.

In small children having persistent symptoms of coryza and nasal discharge a roentgen examination of the paranasal sinuses should be made. Very occasionally one will find a foreign body such as a bean, button or a wad of paper in one of the nasal chambers which the youngster has pushed up into the nose.

Broncholiths act in much the same manner as extraneous foreign bodies in producing bronchiectasis. So little is known regarding the etiology of these calcareous deposits in the lung that little or nothing can be said regarding their prevention. Fortunately broncholiths as a cause of bronchiectasis is apparently not of common occurrence and the relationship, therefore, is of more or less academic interest.

Occluding secretions within a bronchus resulting in bronchiectasis are of considerable clinical importance. Probably the best known example of this kind is postoperative atelectasis. Bronchial occlusion by secretions may also occur in pneumonia, during an attack of asthma or in the newborn from the aspiration of mucus or meconium. While the resultant atelectasis in these conditions can often be suspected clinically, roentgen examination is usually required for confirmation of the diagnosis.

Prophylactic measures for postoperative atelectasis are, at the same time, preventives of bronchiectasis. Frequent change in the position of the patient after operation, the avoidance of excessive sedation which tends to suppress the cough reflex and the stimulation of respiration by carbon dioxide inhalations are measures which have been successfully used to prevent this type of atelectasis. If the atelectasis does not clear promptly bronchoscopic aspirations should be resorted to.

In the presence of atelectasis due to occluding secretions of whatever origin, prompt removal of the secretions by means of the bronchoscope should be carried out. When the roentgenogram in pneumonia indicates a definite atelectasis bronchoscopic aspirations should be considered, provided this procedure is not contraindicated by the patient's condition. In the newborn direct suction by way of the upper respiratory tract will usually be sufficient to dislodge the obstructing material.

Bronchial obstruction may result from a lesion having its origin in the wall of the bronchus itself. In this category are strictures which may be either traumatic or infectious in origin. A traumatic stenosis may result from lodgment of a foreign body or from attempts at its removal. Thus great skill and care must be exercised in the bronchosopic removal of foreign bodies. Prevention has already been considered for strictures resulting from bronchial infection. In stenosis of specific origin, such as occurs in tuberculosis and syphilis, the primary disease is usually more important than any complicating bronchiectasis. The same holds true for the extensive pulmonary fibrosis occurring in tuberculosis and pneumoconiosis. Bronchiectasis oc-

curring under such conditions is usually of secondary importance.

We may here consider all forms of tumors regardless of whether they arise from the wall of a bronchus or press upon the bronchus from without. Such lesions are of frequent occurrence and the malignant varieties are increasing in incidence. There is, of course, no known means of preventing neoplasms. Much, however, can be done for benign adenomata which are much more frequent than generally supposed. Roentgenologically, it is not possible to distinguish between the benign and malignant bronchial neoplasm. All such cases should have a biopsy taken from the tumor by the bronchoscopist. Often the entire tumor can be removed bronchoscopically with complete cure. When this cannot be done and the tumor is histopathologically benign a cure can be obtained by lobectomy. Simply because the vast majority of bronchial tumors are malignant should not lead one to the conclusion that all of them are malignant. Occasionally such tumors are not neoplasms at all but granulation tissue from various causes. Here again the bronchus can be relieved of its obstruction by the bronchoscopist and bronchiectasis prevented.

Bronchial obstruction may occur as the result of pressure extrinsic to the bronchus itself. Enlarged lymph nodes usually result from pulmonary infections the prevention of which has already been discussed. Tuberculosis is a common cause of hilar lymph node enlargements. The prophylaxis of this condition is common knowledge and need not be considered here. Enlarged lymph nodes of malignant origin either primary or secondary and those resulting from the various lymphoblastomata cannot be prevented.

The prevention of bronchiectasis due to pulmonary fibrosis means the prevention of the cause of the fibrosis. The fibrosis of pulmonary tuberculosis has already been discussed. In the fibrosis of pneumoconiosis much has already been accomplished in the prevention of the inhalation of harmful dusts and such efforts are being continued by those interested in this important pulmonary disease. Other types of pulmonary fibrosis are usually of infectious origin and prophylactic measures must be directed toward the prevention of these specific infections.

Finally, under the heading of miscellaneous causes of bronchiectasis are lesions of the pleura and of the thoracic cage. Such conditions are usually secondary so that preventive measures must be directed toward the primary condition. Since these are so varied and numerous and since bronchiectasis from such causes is not of frequent occurrence, an attempt to discuss prevention in this group of cases would serve no useful purpose.

In general, it may be said that two of the most useful and important procedures in the prevention of bronchiectasis are the roentgen examination of the chest and the bronchoscopic examination. In every affection of the respiratory tract in which symptoms are persistent or do not readily respond to treatment, a roentgen examination is indicated, including the sinuses as well as the chest. This will frequently reveal conditions which, if promptly taken care of, will prevent the development of bronchiectasis. This type of examination is now generally available to everyone regardless of how small the community.

More frequent use of the bronchoscope in persistent disease of the respiratory tract would do much toward preventing bronchiectasis by the early detection of those conditions known to give rise to bronchiectasis. Unfortunately this type of examination is not as generally available as might be desired. Adequately trained men in this specialty are still scarce. It is unwise to entrust a patient to a physician who has not been thoroughly trained in this field. It is far better for the practitioner to send his patient to one of the many clinics where expert bronchoscopy is available.

Finally, in the prevention of bronchiectasis an educational program for both the laity and the medical profession is a matter

of prime importance. Laymen must be told and impressed with possible seriousness of the common cold, especially in children. They must be taught the very simple preventive measures available to all. They must have impressed upon them the necessity of seeking medical attention when suffering from repeated respiratory tract infections or when such an infection persists and does not readily respond to the usual remedies. Much can be done in the education of parents regarding the dangers of foreign bodies gaining access to the respiratory and alimentary tracts. In an analysis of 1,135 cases of foreign body in the air and food passages, Jackson and Jackson[17] came to the conclusion that carelessness was responsible for 87 per cent of the cases. Here is a fertile field for education and preventive efforts. Such educational efforts will fall largely to the lot of the general practitioner, the pediatrician and the otolaryngologist who see patients when the etiological factors of bronchiectasis are at work and at a stage when bronchiectasis can be prevented.

Of greater importance at the moment is the education of the medical profession itself. There are so many physicians who know so little about bronchiectasis. There are many others to whom the thought of the prevention of bronchiectasis has never occurred. Physicians must be disabused of the idea that nothing can be done to prevent bronchiectasis. A clearer knowledge of the entire problem of this disease, particularly its etiology and economic importance, will do much to arouse interest of the general medical profession. This educational program must include the roentgenologist because there are many such specialists who do not have the true concept of bronchiectasis and the problem it presents.

In this educational program the roentgenologist has an excellent opportunity to render real service. As has already been stated, he has an opportunity to view the entire life history of bronchiectasis from the etiological stage to the terminal events. He can educate his colleagues regarding many of the pertinent facts of bronchiectasis he can direct their attention to the possibility of a developing or the existence of an early bronchiectasis, and finally he can suggest a bronchoscopic examination when in his opinion this procedure is indicated. Thus, in the prevention of bronchiectasis, a roentgenologist who is conscious of and has a full knowledge of this disease is an asset of prime importance.

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PULMONARY AND OSSEOUS MANIFESTATIONS OF TUBEROUS SCLEROSIS, WITH SOME REMARKS ON THEIR PATHOGENESIS*

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THE protean character of tuberous sclerosis and other ectodermoses makes difficult a satisfactory comprehension of the pathologic process involved. The true nature of some manifestations encountered in this disease, especially in its "form fruste," may remain unrecognized by the clinician, and be misinterpreted by the roentgenologist, unless they are familiar with the relation of these disorders to the ectodermoses. It may therefore not be amiss to report a case studied at the University Hospital, and raise some questions, which, then considered on a larger material, may throw additional light on our knowledge of this interesting syndrome.

CASE REPORT

V.G. (Hosp. No. 77335), white male, aged twenty-five, was admitted to the University Hospital in October, 1934, complaining of polyuria, nocturia, pain in the chest, back and in both legs. For the last two years previous to admission, the patient has been drinking large quantities of water, and correspondingly his urine output has averaged approximately 10 liters daily. The patient had no headaches or visual disturbance. The pain in the chest was dull; patient coughed occasionally, but raised no sputum, and had no hemoptysis. The temperature was apparently normal. The pain in the back and legs seemed to be of muscular character, more intense at night and in the morning, with some improvement after moderate exercise. Because of definite pulmonary pathologic condition, revealed by roentgen examination, the patient was for some time under observation in a tuberculosis sanatorium, but discharged without advice to continue treatment when repeated sputum examinations failed to reveal tubercle bacilli.

On physical examination the patient appeared older than his age; all teeth were absent; no abnormal findings were elicited within the chest or other internal organs. The skin was dry, slightly atrophic, without any abnormal pigmentation.

Roentgen examination of the chest showed an irregular honeycomb pattern, with numerous multangular areas of increased translucency, varying somewhat in size, and separated from each other by rather thick fibrous strands, which formed a nodular arrangement at points of their intersection. There were larger thin walled cystic cavities in the right lung field, without demonstrable fluid levels. The mediastinum was free. There was no displacement of the cardiac shadow toward either side.

Roentgenograms of the maxilla and mandible were reported to have shown a complete absence of teeth, and marked decalcification of these bones, which was suggestive of disturbed calcium metabolism. The remainder of the skeleton was not examined at that time.

After three weeks of observation the patient was discharged, but returned shortly thereafter, stating that one week after his dismissal he had had severe pain throughout the left chest and severe dyspnea. The pain has continued, and the patient was raising large amounts of watery material.

On physical examination signs of pneumothorax and mediastinal displacement were elicited. Otherwise the patient presented the same features as on the previous examination.

Roentgen examination of the chest showed pneumothorax of the left pleural cavity, with marked collapse of the left lung, and a contralateral displacement of the heart and mediastinum. There were no structural changes within the lungs not seen on the original examination. A roentgen diagnosis of polycystic lung disease with pneumothorax subsequent to a rupture of one or more of the small cysts was made.

The patient remained under clinical observation, in the course of which the skeleton was examined roentgenologically, and the blood chemistry was studied.

* From the X-Ray Department of the State University and Crippled Children's Hospitals (John E. Heatley, M.D., Director)

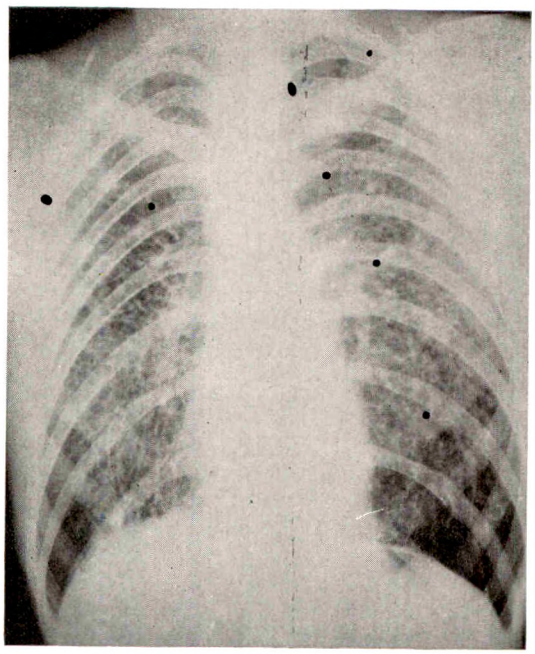


FIG. 1. Both lung fields show a honeycomb pattern with numerous multangular areas of increased translucency, separated by thickened fibrous strands.

Roentgenograms of the vertebral column showed an asymmetry of the body of the first lumbar vertebra, whose left lateral margin was slightly excavated. On the lateral view there was a fairly large erosion of the anterior portion of the vertebral body. The bone adjacent to this erosion was moderately sclerotic. Both vertebral plates were preserved. Within both iliac bones there were several large, sharply defined areas of rarefaction, varying considerably in size and shape. The adjacent bone trabeculae were of increased density, coarse and somewhat irregularly arranged. The medial portion of the wing of the left iliac bone, near the sacroiliac articulation showed numerous smaller cyst-like areas of decreased density, with definite evidence of confluence of several of these cysts. There were similar areas of rarefaction within the right pubic bone, extending even beyond the cortical portion of the bone. A few fairly large bone spicules extended from the pubic bone toward the foramen obturatum. Both femoral necks and the juxtatrochanteric portions of the shaft showed numerous cystic areas, varying in size from that of a hazel nut to that of a large hen's egg. Surrounding these rarefactions was usually a narrow zone of sclerotic bone. The shafts showed slight expansion at the sites of the cystic cavities. The diaphyses of both femora showed a diffuse fibrillation of the cancellous bone, with some thickening of the bone trabeculae, which were fairly separated by fusiform areas of rarefaction. The cortex remained intact.

The blood count showed no deviation from normal. The blood calcium was 9.8 mg. per 100 cc.; phosphorus 4.2 mg., phosphates 3.56 mg. The nonprotein nitrogen was 35 mg., uric acid 3.5 mg., blood sugar 80 mg. and cholesterol 133 mg. per 100 cc. The daily amount of calcium excreted in the urine on a calcium free diet was 528 mg.

In May, 1940, the patient was readmitted to the hospital. At this time he complained of polydypsia, polyuria and pain in the back and both lower extremities. He had no complaints referable to the chest.

In addition to the findings previously mentioned, there was marked muscular atrophy, especially of the lower extremities; flexion contracture of 10 to 15° of both thighs and flexion contracture of 10° of the right knee, and 20° of the left knee. There was some tenderness over the right thigh posteriorly, close to the

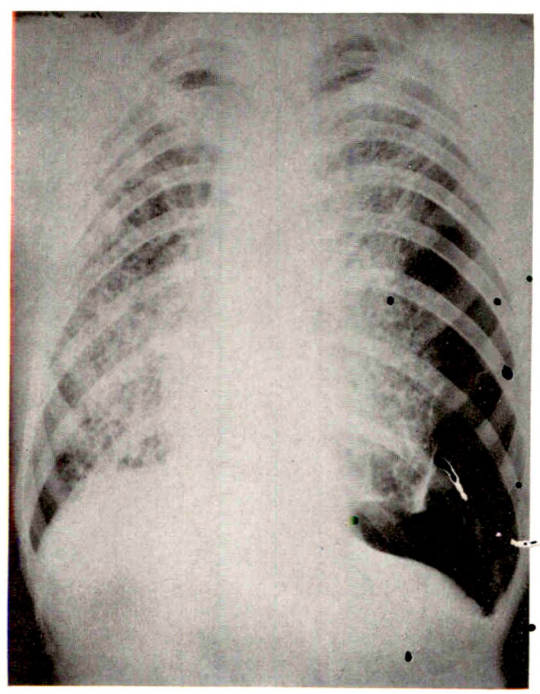


FIG. 2. Pneumothorax reveals numerous small subpleural cysts.

greater trochanter. The spine showed a marked prominence and slight right scoliosis of the twelfth dorsal and first and second lumbar vertebrae. There was some tenderness over this segment of the spinal column.

A re-examination of the skeleton and chest showed no appreciable progress of the pathological changes within this period of time.

From the patient's history and clinical findings, it was obvious that he presented a definite diabetes insipidus, polycystic lung, and a certain form of multiple osteodystrophy.

In absence of signs of an endocranial tumor over an eight year period of observation, it seemed safe to conclude that the diabetes insipidus was not caued by a neoplasm of the pituitary body. The blood chemistry did not support a possible diagnosis of osteitis fibrosa cystica due to hyperarthyroidism. The blood calcium and phorphorus level were found normal. The pulmonary pathologic condition exhibited by the patient was of great interest *per se*.

Attempting to reduce these various clinical complexes to one common etiological factor, we considered the possibility of Schüller-Christian's disease, a systemic disorder in which diabetes insipidus and skeletal alterations are the outstanding features of the disease. Although involvement of the lung is extremely rare, there are known cases of generalized xanthomatosis with miliary dissemination in the lungs. We were unable to find sufficient evidence to warrant this diagnosis in our case. The characteristic cranial defects were absent; there was no demonstrable disturbance of the lipoid metabolism.

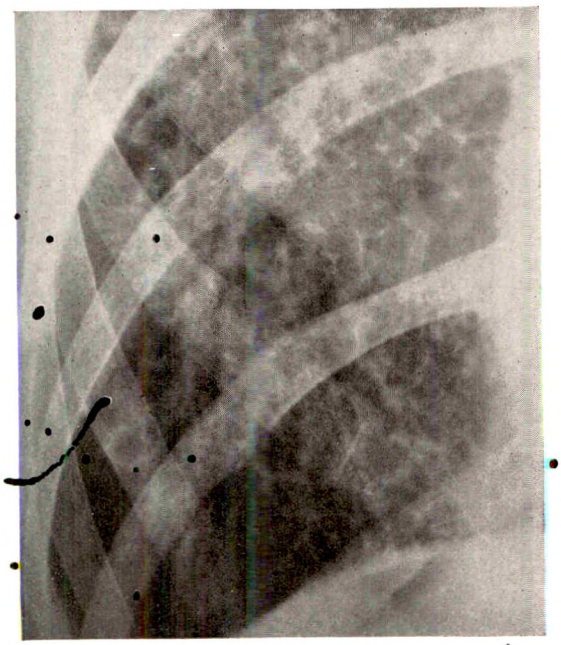


Fig. 3. Basal portion of the right lung demonstrating the detail of the pulmonary pattern.

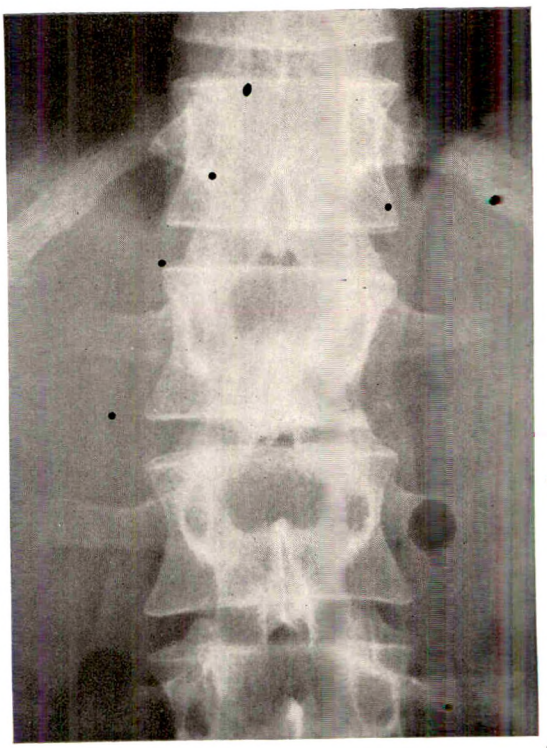


Fig. 4. The lateral margin of the first lumbar vertebra is excavated.

Tuberous sclerosis seemed to us to be the only disease which could explain the manifold symptoms presented by the patient, and account for the patho-anatomical changes in such divergent organs as brain, lung and skeleton.

According to our present conception, tuberous sclerosis is a form of congenital ectodermoses, which in addition include three other outstanding entities: (1) von Recklinghausen's neurofibromatosis; (2) anginomatosis cerebri, and (3) Hippel-Lindau's disease.

A complete discussion of the clinical manifestations of tuberous sclerosis would exceed the scope of this presentation, and we refer to the literature on this subject.

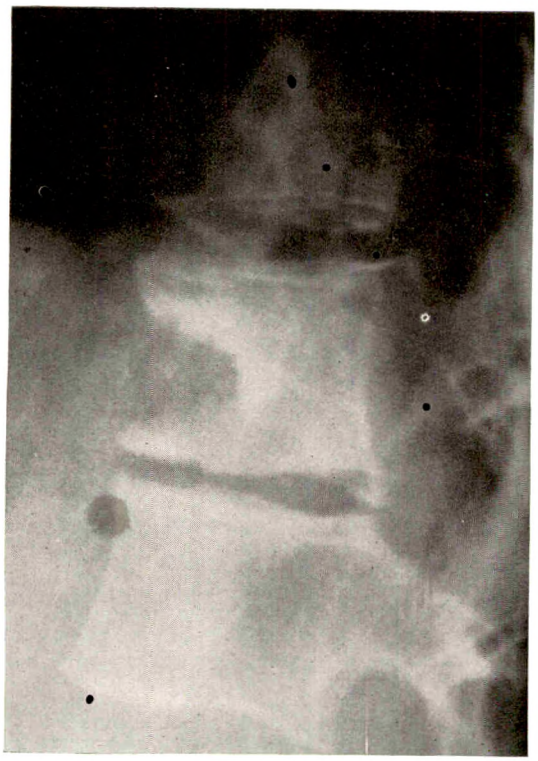


Fig. 5. Lateral view of the first lumbar vertebra shows an erosion of the vertebral body and sclerosis of bone adjacent to it.

In fully developed cases one finds most frequently the following symptoms: (1) epileptic seizures (genuine or jacksonian); (2) mental deficiency; (3) cutaneous lesions, known as adenoma sebaceum, and (4) congenital tumors of the eye and many visceral organs (rhabdomyoma, hypernephroma, renal lipomyosarcoma, tumors of the spleen, lymph nodes, lungs, retina and bones).

Clinical manifestations of the disease, when fully developed, appear rather early and are often recognizable in infancy. The protean character of the disease, which may in some instances present only certain of its characteristic features, quite frequently precludes an early diagnosis unless one is aware of its symptomatology. Many patients afflicted with this disease may present only abortive manifestations, which sometimes become apparent in the later decades of life. Cases in the sixth and seventh decades of life have been reported. The clinical symptoms depend on the organs involved. In some cases the endocranial lesions dominate the clinical picture, in others the visceral tumefactions are responsible for the patient's complaints.

Pathologically, tuberous sclerosis is characterized by the presence of potato-like tumors, varying in size from 0.5 to 3.0 cm. in diameter. These tumors occur most frequently in the cerebral hemispheres, although other parts of the central nervous system may also be involved. In the walls of the third and the lateral ventricles, immediately beneath the ependyma, small irregular projections are found, arising usually from the thalamus. These appear as tiny, button-like tumors, or when larger in size as "candle-gutterings" (Lind) running from above downward and following the tributaries of the vein of the corpus striatum. Although macroscopically the tubera seem to be sharply demarcated, there is no abrupt transition between normal and diseased tissue, when studied microscopically.

The microscopic alterations characteristic of tuberous sclerosis affect the pyramidal cells and the glia. The pyramidal cells may be present in the more superficial layers, and granular elements may be scattered among the larger neurones of the deeper parts. The orientation of the pyram-

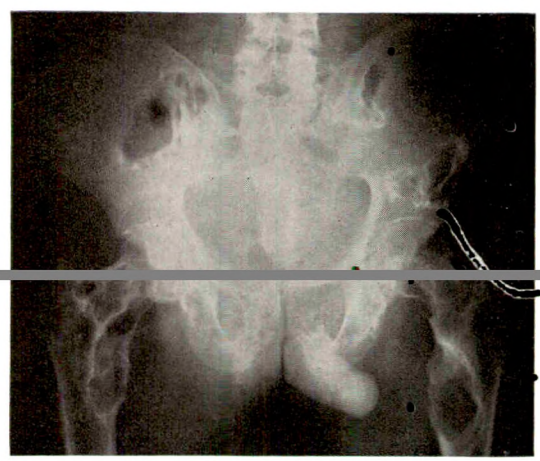


Fig. 6. Numerous cystic areas within the iliac, ischial and pubic bones and both femora.

idal cells may be altered so that these cells assume an abnormal polarity. The morphological state of many of the cells is changed. There may be a clumping and later disappearance of Nissl granules; the nucleus may assume a lateral position. The process may become thicker, and the cells as a whole may skrink.

The neuroglia exhibits marked proliferation. There is a considerable overgrowth of glial fibrils which are mainly of the thin type. The proliferation occurs primarily within the cortex, though it extends for some distance into the underlying white matter. One finds peculiar interlacing of the fibers. In the subependymal growths the neuroglial proliferation produces a loosely interlacing complex of parallel strands. The ependymal covering remains intact, and between this layer and the glial overgrowth is an interval occupied by loose areolar tissue. In the intraventricular growths the glial proliferation may become excessive and give rise to definite, macroscopically discernible tumors.

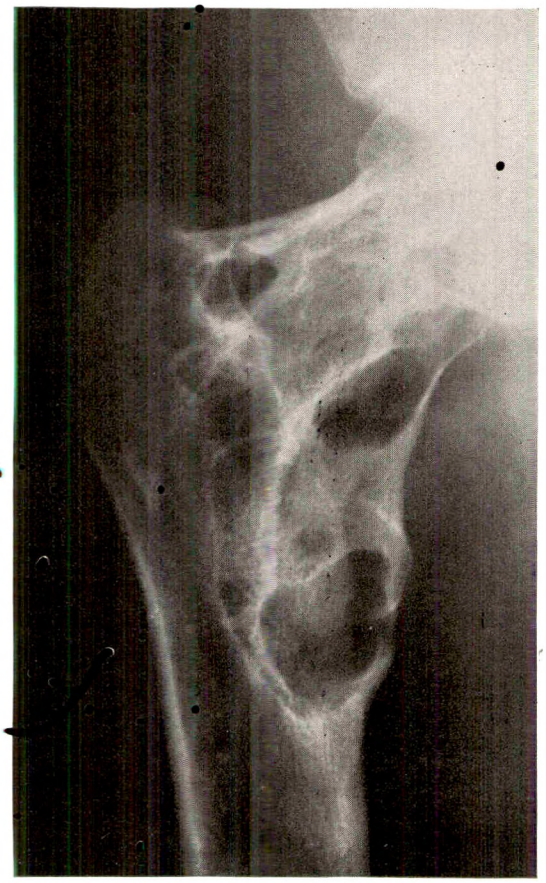


Fig. 7. Detail view of the left femur shows multiple, partly confluent cysts.

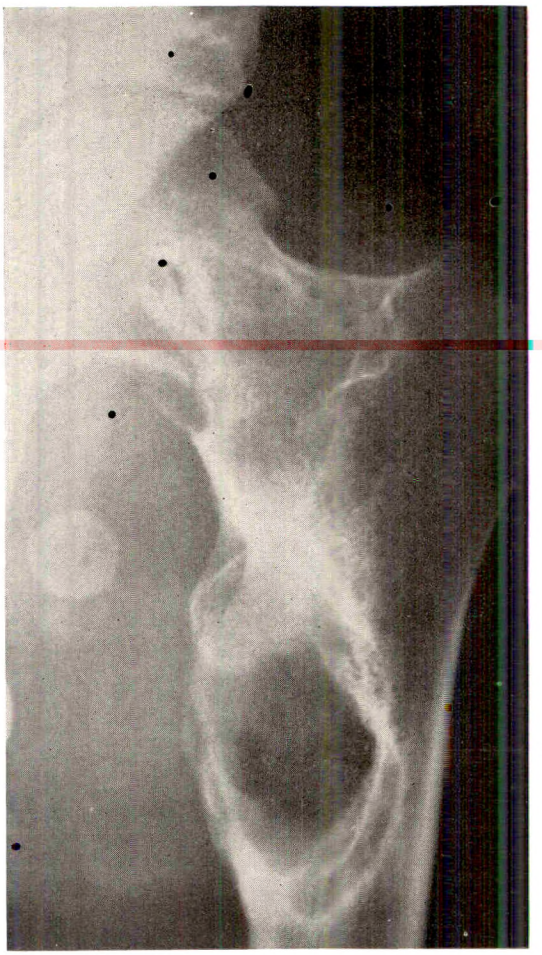


Fig. 8. Detail view of the cystic changes and associated sclerosis of the right femur.

Great significance has been attached to the giant cells within the foci of tuberous sclerosis. Since Harthagen's original description they have been noted by all writers concerned with this subject, and have caused much discussion as to their nature and their bearing upon the conception of tuberous sclerosis in general.

These cells lie typically in the depth of the sclerotic nodules, i.e., where the neuronic and glial changes are most intense.

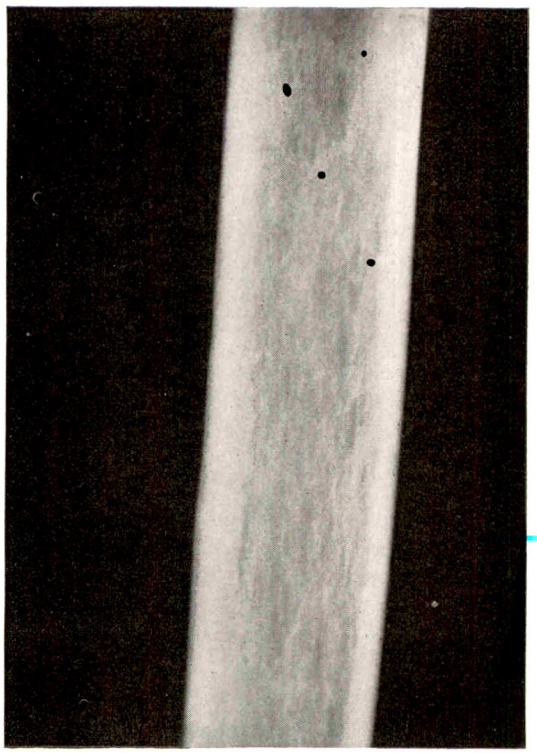


Fig. 9. Shaft of the femur, showing fibrillation of the cancellous bone and fusiform areas of rarefaction.

Both cortical and subependymal tubera may contain such cells, though they are more numerous in the former situation. It is important to realize that they may occur outside of the cerebral cortex; thus they appear as heterotopies in the white matter near the tubera. Furthermore, they may be demonstrable in the cortex away from the sclerotic regions, and they have also been noted in more remote portions of the nervous system such as the cerebellum, medulla, and spinal cord.

Much controversy has arisen as to the nature of these large cells, and in particular as to whether they are derivatives of glial or nerve elements. It appears definite from careful histopathological studies that all the large cells of a particular case do not have the same morphological and staining characteristics and probably represent entirely different cells, their sole common feature being their large size.

A large cell of neuronic type and a large cell with glial resemblance are distinguished.

The large cells usually predominate slightly and are more numerous in the cortical than in the subependymal growths. They are well demonstrated by the ordinary nerve cell stains and particularly by Bielschowsky's method. They do not react to glial stains. They vary in size from a large pyramidal cell up to that of a Betz ganglion cell; and when secondary changes occur, further distention may increase their bulk still more. As a rule, they occur in clusters of three or more and may be surrounded by some densification of the glia. They are occasionally triangular in outline but the larger specimens are elliptical or even circular.

The internal structure of these cells shows various abnormalities in the position and staining reactions of the nuclei. Considerable variability exists in the character of the process; sometimes they are short and thick, and at times a stem arises which quickly breaks up into finer branches. The neuronic character of this type of cell is suggested by the presence of intracellular fibrillary networks and also of Nissl granules.

The large cells with glial resemblance are somewhat less numerous and occur more particularly in the ventricular nodules. They are also grouped in the vicinity of blood vessels, to the walls of which they may be attached. Their shape in unlike that of a nerve cell, being oval or circular. There are no intracellular fibrils or granules, and the processes are invisible except in silver stained preparations. With such methods the processes are displayed, sometimes recalling those of a nerve cell, but at other times numerous short and curly processes radiate at intervals from the periphery.

Histopathological studies of other organs, frequently affected by the pathologic process, underlying tuberous sclerosis and allied conditions, have been extensively reported in the medical literature. Only

observations regarding pulmonary and skeletal involvement are very scant.

Berg and Vejlens observed 3 cases of tuberous sclerosis with pulmonary manifestations, 2 of which occurred in members of the same family. One of these cases has been studied by them anatomo-pathologically and we should like to quote the description of their findings:

> The lungs presented a unique appearance. They had everywhere, except laterally at the right tip, grown on the wall of the thorax by means of rather loose connective adherences. The free space corresponded to a pneumothorax cavity of the dimension of a good sized tangerine, the existence of the cavity having been previously proved roentgenologically. The lungs were large and voluminous and felt to be more air-cushioned than usual. The surfaces were dotted with small air filled bubbles, not more than pea size, suggesting a bullous emphysema. The walls between the larger cysts often reached 0.5 cm. in thickness, but they contained a number of miniature cysts. In places the cyst walls were only the thickness of paper and had been perforated.
>
> Microscopically the walls of the cyst consisted to a large extent of connective tissue containing numerous blood vessels. But the most salient component was plain muscular tissue. This muscular tissue moreover was not, as in the description of cystic lung previously given by Buchmann and Oudendal and others, disposed in thin, superficially parallel layers. Nor did it bear resemblance to the more irregular muscular proliferations of von Steussel's muscular cirrhosis of the lung. It had the unmistakable character of tumor tissue, a fact which differentiates the present "cystic lung" from all other cases described hitherto. The nuclei of the muscular fibers were in the main somewhat broader than usual. Further the contractile substance, which is stained yellow in the normal way by picric acid, according to van Gieson's method, had not always the same regular, narrow, fusiform shape, which is so characteristic of the smooth musculature. The muscular fibers were often shorter and thicker and blunted at the ends. They are probably to be regarded as survivals from the embryonic stage, without however presenting the appearance of malignant tumor cells. The essential characteristic of this myomatous tissue was that the muscle fibers were joined together in intercrossing bunches and clusters, precisely as in the case of ordinary myoma, and in the same way as in the tumor-like formations in the kidney, for example. The lungs presented, apart from the general cystic transformation, the aspect of a diffuse myomatosis, which must be considered as very closely related to the genuine tumor formation.
>
> It is doubtful whether the lung cysts in tuberous sclerosis should per se be characterized as malformation, and if one knows that sclerotuberous alterations were established in the brain and the kidneys, as usual, and were also found in a single lymphatic gland and in the spleen, and if, further, due regard is paid to the fact that the disease is of familial character, then one is justified, it would seem, in holding that the myomatous changes in the lung tissue are also an expression of a general malformational disease with a tendency towards tumor formation.

Roentgen studies of these cases were reported by Berg and Zachrisson, and they conform with those observed in our case. These authors emphasized the irregular, honeycomb-like pattern made up of scarcely visible to hazel nut sized translucencies, separated by fine, round striae, noticed in both lung fields. These markings seemed to become less dense at the apex, where the cavities were of larger size. The points of intersection in the honeycomb structure did not show any particular tendency to mottling, although the appearance on the whole resembled that seen in different forms of miliary dissemination. They discussed the differential characteristics between the tuberous sclerosis and other miliary disseminations.

Skeletal involvement in cases of tuberous sclerosis has been reported by Gottlieb and Lavine, Kveim, and Heublein, Pendergrass and Widmann.

The histopathological alterations of the skeleton have been described by Kveim, according to whom "in the decalcified sections one finds cancellous bone, with thick bone trabeculae. The bone architecture is

irregular; it looks more like cartilage and shows a poor calcium content. Few cells are visible within the marrow spaces of the bone; instead one encounters, scattered here and there, diffuse, fibrous tissue, poor in cells. No signs of inflammation or tumor formation are present. From the periosteum toward the bone one finds nearly homogeneous, hyalinized connective tissue clumps and masses. Though signs of inflammation are absent, the marrow shows isolated areas, resembling osteitis fibrosa."

The roentgenological findings observed within the skeleton by these authors show a marked similarity.

Gottlieb and Lavine found periosteal thickening and generalized fibrosis of metatarsal and metacarpal bones and their associated phalanges. There was also a generalized fragmentation of the cortical layers. Several of the bones of the hands showed areas of marked rarefaction, suggesting small cysts. The bones of the cranium showed a peculiar mottling, with indistinct islands of increased density alternating with areas of rarefaction throughout the bone. Both the internal and external tables appeared thicker and denser than normal.

Kveim reported in his cases the presence of similar changes affecting the cranial bones. The bones of the hands showed periosteal thickening, sclerosis and centrally located areas of rarefaction within the phalanges and metacarpals. The skeletal changes resembled those of osteitis fibrosa.

The presence of tumors and other disorders within organs derived from the mesoderm, associated with tuberous sclerosis, which is preëminently a disorder of the ectodermal layer, caused numerous theories to be advanced, which attempted to place all these disorders on a common basis.

Bielschowsky was of the opinion that the presence of cutaneous and visceral lesions in close association with tuberous sclerosis was of utmost importance. All of these alterations, in his view, could and should be reduced to congenital, or even better, embryonal developmental disturbances. The manifestations of this disturbance are not uniform. Whereas in one organ the element of developmental inhibition predominates, in other organs are observed enormous new formations, which apparently have their origin in a faulty blastomatous developmental tendency of some tissue components. These changes occur most likely in early embryonal life, but they do not terminate with birth. A formation of new foci probably does not occur in extra-uterine life. The preformed foci are, however, capable of further growth.

Kveim thought that none of the ectodermoses is a genuine neoplasm, but that the condition is due to anomalies of fetal differentiation, so-called "embryo-plasmatic dysplasias," which chiefly attack the ectoderm—first and foremost the neural plate—at a very early stage. Kveim also considered a possible genetic connection with Recklinghausen's neurofibromatosis, in which he assumed that there also exists a fetal dysplasia of the ectoderm-mesoderm (the neural plate?). While the cell elements affected here are essentially those which form the origin of the sheaths of Schwann, so that neurinomas are localized in the peripheral nervous system ("spongioblastosis peripherica"), the cell elements chiefly impaired by tuberous sclerosis are those which in the fully grown individual are localized in the central nervous system ("spongioblastosis centralis"). Kveim explained the occurrence of tumors derived from mesodermal structures on the basis of polyvalent possibilities of differentiation of all cells at a very early stage of fetal life. The connective tissue elements may therefore be looked upon as originating from the same cell elements as the epithelial part of the tumor. The dysplastic cells are characterized by retarded differentiation.

With regard to the multifarious tumors, consisting partly of cells, difficult to classify, occurring in the internal organs, derived from the mesoderm, he states that these have developed by the same "embryoplastic dysplasia," certain cell elements having been attacked before any differen-

more, increased or diminished production may follow a central nervous disorder. Thus, hypophysectomy in dogs and rats is followed by regressive changes in the parathyroids (P. E. Smith, S. Koster and A. Geesink) and in the toad leads to hypocalcemia (H. A. Shapiro and H. Zwarenstein). These facts throw light on the following observations: A woman, 52 years of age, with long-standing diabetes insipidus, developed signs of Simmonds' disease and a hypocalcemic tetany; a man, 38 years of age, having suffered repeated head injuries by beating, developed a chronic brain lesion, revealed by pneumencepahlography as atrophy and hypocalcemic tetany.

The cases of precocious puberty with osteopathy lack all evidence of an increased production of parathyrotropic hormone and of parathyroid hyperactivity. There seems no other way in which to interpret their mechanism than to assume that the hypothalamic lesion, while provoking other phenomena via the anterior pituitary, increases the sensitivity to the normally produced parathyroid hormone.

The influences emanating from the central nervous system and particularly from the hypothalamus determine the hypersensitivity as well as the resistance of the effector organs. If a pathologically increased quantity of hormone is taken up by more susceptible, but dimensionally limited areas of the bony substance, a stormy reaction will set in, with large amount of lime salts released and with vehement local changes, which lead to the development of 'brown tumors'. When the same quantity of hormone is distributed over the entire skeleton the resultant pathologic changes will develop more slowly, and will be less intense. . . . In analyzing the findings one must consider that the osteopathy, irrespective of whether the disease leads to cyst formation or to generalized osteoporosis, does not, as a rule, advance steadily. There are periods during which the cystic osteopathy is arrested and remains stationary, so that at times the blood calcium level is found to be normal or only slightly elevated.

Perhaps not the neuro-endocrine factor alone may be responsible for the bone changes, which resemble somewhat those described by Freund in osteodystrophia fibrosa unilateralis.

Freund refers in his article to the experimental work of Bentzon and concludes that these skeletal changes may be due to a disturbance in the innervation of the vessels. They would then represent a reaction to active hyperemia of bone tissue. The hyperemia is due to anomalies in the vegetative nervous system, followed by paresis of the muscles of the vessels with vascular dilatation. It may well be that the neurovascular changes play an important part in producing fibrous dystrophies of bone.

The consideration of the pathophysiological aspect rather than the anatomopathological aspect alone seems to us to be of great importance. We so often look upon the mature skeleton as a static organ and lose sight of the anabolic and catabolic processes taking place within the bones. These processes are dependent upon neuroendocrine and neurovascular influences through a mechanism of mutual correlation. The activity of the parathyroid glands is controlled by the parathyrotropic hormone, the production and discharge of which is linked to the metabolism of calcium and phosphates. The parathyrotropic hormone does not seem to be released directly, but is stimulated through the hypothalamic center, which is specifically sensitive to calcium and phosphate concentrations. On the other hand, a lesion within the hypothalamus may increase the tissue sensitivity to normally produced parathyroid hormone. If anomalies of the vegetative nervous system cause also paresis of the muscles of the vessels and vascular dilatation, then the metabolic process within the bone is even more impaired. As a result of these abnormal functional phenomena, the bones show marked alterations of their anatomical structure. Because of the histopathological picture of fibrosis, they are classified as fibrodystrophies.

We have presented these views regarding bone changes in tuberous sclerosis because they offer a functional explanation of the involved process, and may form an important link in the understanding of other bone dysplasias in which one does not find an obvious disturbance of the metabolism of calcium and phosphates. Whether or not

tiation of the blastula into the ectoderm and mesoderm has taken place. The several strange cell forms of these tumors may indicate also a kind of subsequent differentiation process.

Worster-Drought, Dickson, and McMenemey also turn to changes effected in early embryonal life for an explanation of the occurrence of ectodermal and mesodermal tumors in any of the neurocutaneous syndromes. They call attention to the experimental work of Needham, Waddington, and their associates on the so-called "embryonic induction." According to this hypothesis "evocator substances" are released by the nerve fibers, which call forth normally a parallel arrangement of the Schwann cells and the orderly distribution of the adjacent mesodermal supporting tissues. Any derangement affecting the nerve elements (ectoderm) and, therefore, the evocator substance, may gravely influence the surrounding mesoblast, producing such changes as are encountered in neurofibromas, multiple meningiomas, etc.

Gottlieb and Lavine, Kveim, and Yakovlev attributed the skeletal changes to the developmental disorder of the mesoderm, and Yakovlev, in view of the accepted affinity of tuberous sclerosis and neurofibromatosis, considered the possibility that the cystic cavities within the bones may be due to true Schwann cells arising from the periosteal nerves, and destroying the neighboring bone by pressure rather than invasion.

A close study of the osseous changes in tuberous sclerosis reveals that they are not uniform. One finds bone sclerosis, periosteal thickening, bone rarefaction, and cyst formation. Very few of the cysts could be classified as of subperiosteal origin, and one does not find the sharp line of demarcation usually associated with pressure erosion.

The histopathological changes, resembling those of osteitis fibrosa cystica, do not contribute to our understanding of them, since this histopathological picture is a too frequent response of bone marrow to different damages and stimuli. It may occur in a number of essentially different bone diseases, such as inflammatory, neoplastic, metabolic and simple mechanical disturbances, so that the histopathological diagnosis of osteitis fibrosa quite often may, in the special case, be more obscuring than revealing. In a great number of cases, it is only a symptomatic and not a nosologic diagnosis.

The pathogenesis of the skeletal changes in tuberous sclerosis, and possibly also in neurofibromatosis, may perhaps be better understood if one associates them with those in the central nervous system and sees in them an expression of physiopathological disturbance rather than of embryonal tissue dysplasia.

The cases exhibiting skeletal changes showed the tubera to be located in the floor of the lateral ventricles and within the third ventricle. In other words, the lesions involved the thalamic and hypothalamic area of the brain. We may also assume that in our case the diabetes insipidus was due to involvement of this area.

The significance of the hypothalamus in skeletal disorders has recently been emphasized by Lichtwitz, who in a discussion of bone pathology of hypothalamic origin states:

> Although no case has been reported in which such a central nervous lesion [destructive processes in the hypothalamus] produced precocious sex maturation and cystic osteopathy simultaneously, there is sufficient evidence to prove that bone disorders develop following encephalitis. . . . The history of a man, who at the age of 41, fell ill with a grippe which was characterized by sleep inversion, is of particular interest in this connection. After the illness, he developed the following features: diabetes mellitus, hepatosplenomegaly, atrophy of the gonads, arthritis and a generalized osteoporosis with many large cysts in both humeri and femurs. . . . Spontaneous fractures occurred repeatedly. For the last 2 years the bone disorder has remained stationary. A blood chemistry at this time showed 10.6 milligrams per cent calcium and 2.0 phosphorus . . . .
>
> In this case a hypothalamic lesion brought on —beside other disorders— cystic bone disease. . . . As regards the parathyrotropic hor-

this conception can be sustained will depend largely on observations made on a larger material of the same type.

Concerning the pulmonary alterations we are unable to offer any original views, and hold with the other authors that they are an inherent manifestation of the basic disorder underlying tuberous sclerosis, i.e., an expression of an "embryoplasmatic dysplasia."

SUMMARY

1. A case of tuberous sclerosis with associated pulmonary and skeletal changes has been presented.

2. The polycystic lung disease is considered to be an expression of "embryoplasmatic dysplasia."

3. The osseous manifestations may represent functional alterations due to neuroendocrine and neurovascular disorders, caused by the involvement of the hypothalamus and the vegetative nervous system.

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* See for other references.

A NEW CHOLECYSTOGRAPHIC PREPARATION:

By H. C. OCHSNER, M.D.*
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A RÉSUMÉ has been made of the results of 300 consecutive cholecystographic studies made during a period of three months, using a new contrast medium. The preparation, beta (4-hydroxy-3,5 diiodophenyl) alpha phenyl propionic acid, is supplied in tablet form and its administration is consequently simpler than that of tetraiodophenolphthalein. I have been particularly interested in the reactions experienced by patients who have taken this preparation and am firmly convinced that toxic reactions are less frequent and severe than with other contrast media; this without sacrifice of accuracy.

One hundred and sixty-nine, or 56 per cent, of the patients examined noted absolutely no untoward effects, and an additional 58 experienced slight reactions, making a total of 227, or 70 per cent, who had slight or no symptoms of toxicity. Reaction was moderate in 60, or 20 per cent, of the patients and severe in only 13, or 4 per cent.

Although nausea occurred in 70, or 26 per cent, of all cases, it was mild in 52, or two-thirds of this group. Nausea was associated with vomiting in only 8 patients, or less than 10 per cent of the group in which it occurred. In other instances, there was associated diarrhea, abdominal pain, pain on urination, or both. Diarrhea occurred in 46, or 15 per cent, of the total group; in most cases it was of slight or moderate severity. Abdominal pain was a complaint of 28, or 9 per cent, of the patients studied; it was severe in only 2 instances. Sixteen, or 5 per cent, of the patients complained of pain in the bladder area or pain on urination; this was slight in 5, moderate in 10, and severe in only one.

There was excellent, good or fair visualization in 201, or 67 per cent, of the total group and, of these, stones were demonstrated in 19, or 9 per cent. There was poor visualization in 93, or 31 per cent, of the total; stones were demonstrated in 39, or 42 per cent, of this number.

That intolerance to the medium was not the cause of the failure of satisfactory visualization of the gallbladder would appear to be proved by the fact that fewer reactions occurred in the poorly functioning or non-functioning group than in the group in which satisfactory visualization was obtained. Reactions were more severe in the patients who had demonstrable stones. In the normally functioning group without stones, 33 per cent of the patients had reactions of moderate or marked severity, while in the same group with stones, 47 per cent of the patients had reactions which were moderate or marked. In the non-functioning group without stones, only 7 per cent of the patients experienced moderate or severe reactions, while of this group in which stones were demonstrated, 23 per cent had similarly severe reactions.

Only 23 of these patients have been subjected to surgery but no error occurred which could be attributed to the method. In this series there has been no instance in which operation has been performed in a patient with "normally functioning gallbladder without stones." In 3 instances, in which diagnosis was made of "normally functioning gallbladder with stones," pathological findings were cholecystitis with stones. Two patients were explored who had "poorly functioning" gallbladders without demonstrable stones and in each chronic cholecystitis with stones was found. Diagnosis of "poorly functioning gallbladder with stones" was confirmed in 4 instances; evidence of cholecystitis was

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found and stones were demonstrated. The accuracy of the diagnosis of "non-functioning" gallbladder was established in 14 patients. There was only 1 error and this cannot be attributed to the method. In the absence of a visualized gallbladder in this case, diagnosis of the presence of a small stone was incorrect but there was definite evidence of chronic cholecystitis. In 6 of the 7 patients who had "non-functioning" gallbladders in which stones were not demonstrated roentgenographically, stones were found by the surgeon. In each of 7 others with roentgenographically demonstrable stones, this finding was corroborated surgically.

SUMMARY

Priodax (Schering), beta (4-hydroxy-3,5-diiodophenyl) alpha phenyl propionic acid, a new preparation for cholecystography, has proved to offer many advantages over preparations previously in use. It is reliable, easy to take and produces fewer reactions than other cholecystographic media we have used. There is little or no residue of the medication in the bowel, because this substance is excreted largely through the kidneys, and this eliminates bowel shadows which are sometimes confusing. The gallbladder visualization which is produced is of ideal density to permit the demonstration of stones if they are present.

CHOLECYSTOGRAPHIC STUDIES WITH PRIODAX

By MAX DANNENBERG, M.D.
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THE discovery and development of cholecystography by Graham and his coworkers have been largely responsible for the great advances made in the study of the function and diseases of the gallbladder. Today the technique of roentgenologic examination of the gallbladder has been fairly well standardized.

The cholecystographic products on the market up to now have all been basically the sodium salt of tetraiodophenolphthalein under various trade names. It was first administered only by the intravenous route. However, because of the large number of severe reactions, Graham and his colleagues began to experiment with the oral method. Nausea and vomiting occurred to a lesser degree. If the patient vomited or a shadow of the gallbladder was not produced, the findings were confirmed both orally and intravenously. The oral method is the one of choice because of the convenience, both to the doctor and patient, the greater safety and the decreased number of reactions. The results approximate the accuracy of the intravenous method, as noted by Beilin and Carlson,[1] Kirklin,[3] and Robinson.[6]

Although the number of toxic reactions with sodium tetraiodopherolphthalein is considerably less by the oral method than by the intravenous, they occur often enough

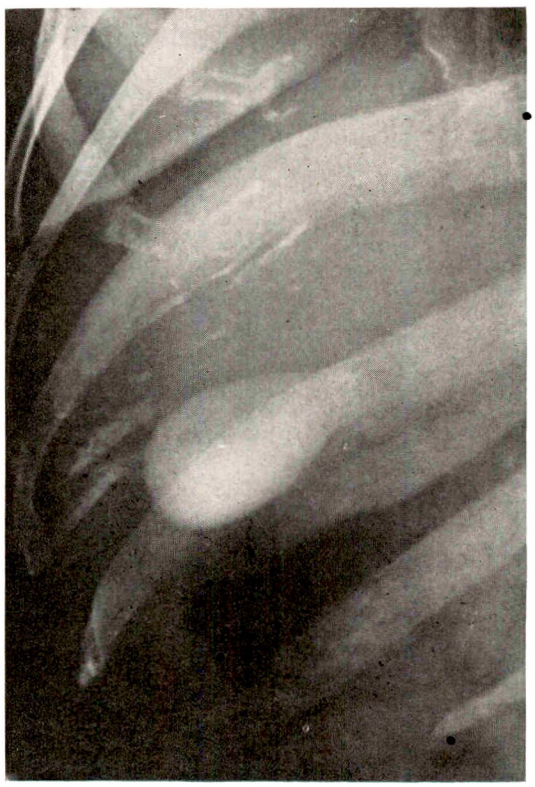


FIG. 1. F.F. Good concentration of contrast medium within the gallbladder. Note absence of the medium in the colon.

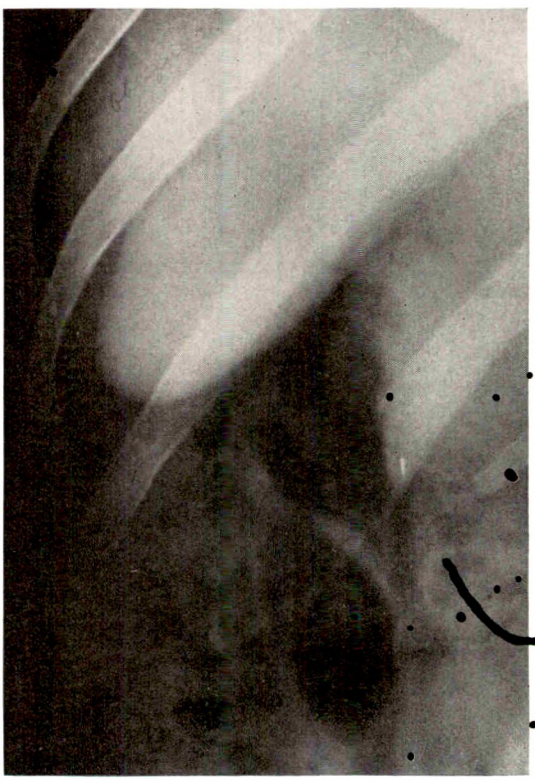


FIG. 2. J.S. Good concentration within the gallbladder. Oblique view throwing the gas shadows away from the fundus.

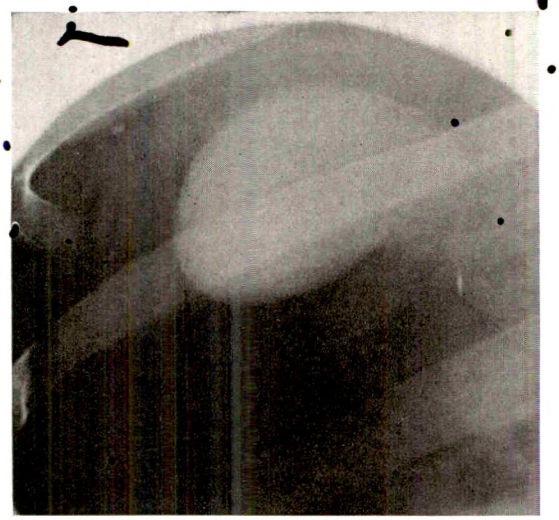


Fig. 3. D.T. Weight, 269 lb. Double dose of priodax. Good concentration within the gallbladder.

to interfere with the efficacy of this drug. As we all know, the sodium salt of iodophenolphthalein is poorly tolerated by many patients. Severe reactions of vomiting, diarrhea and abdominal cramps are often experienced. Golden,[2] Levyn, Aaron,[4] and others, attempted to prevent gastrointestinal reactions by the use of fruit juices, alkalies and paregoric before the administration of the drug. The toxicity of the halogenated phenolphthaleins is due chiefly to the phenolphthalein part of the molecule. In addition to the toxicity, the laxative action of the phenolphthalein

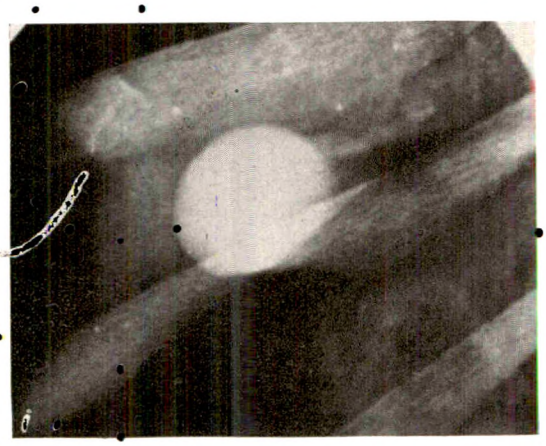


Fig. 4. Good concentration and normal visualization.

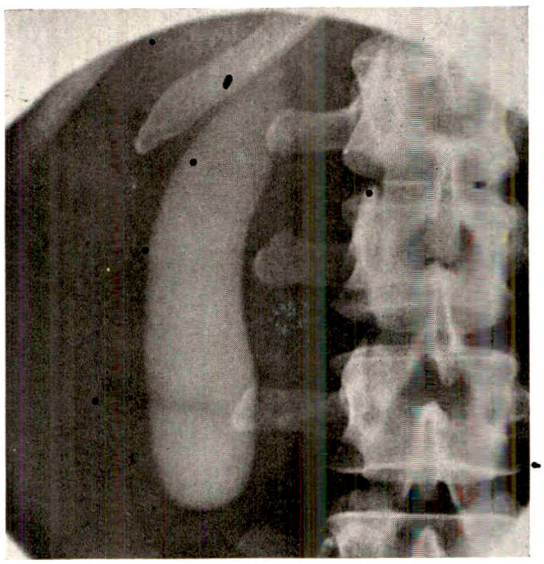


Fig. 5. Good concentration within the gallbladder. Note absence of contrast medium within the colon. Homogeneity of shadow.



Fig. 6. Medium sized gallbladder. Absence of contrast medium in the colon aids in identifying the gallbladder.

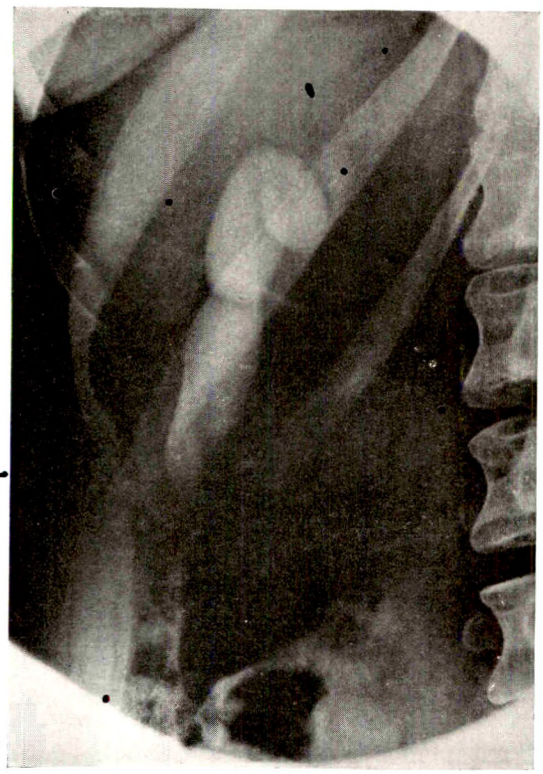


FIG. 7. Good concentration and even density of the gallbladder. There is a constriction in the mid-portion of the fundus. Operative findings: Pericholecystic adhesions with a kink in the middle of the fundus.

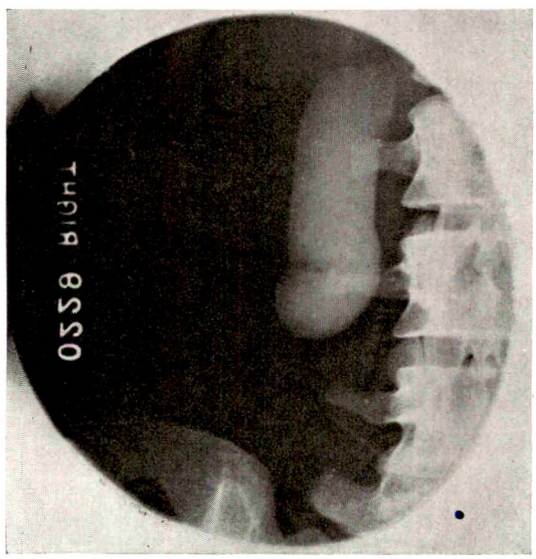


FIG. 8. Medium sized gallbladder of good concentration. There are numerous small, faceted, vacuolated areas which are characteristic of cholelithiasis.

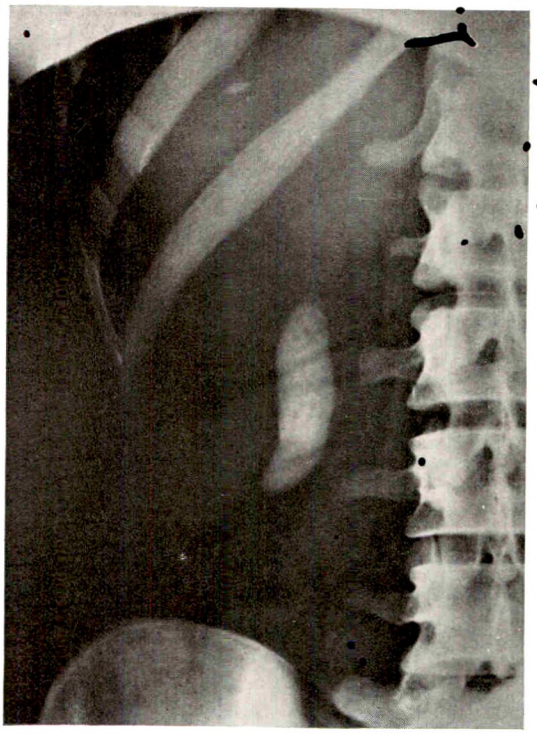


FIG. 9. Fatty meal. The small, faceted, vacuolated areas are clearly visualized. There is a moderate evacuation of the contrast medium.

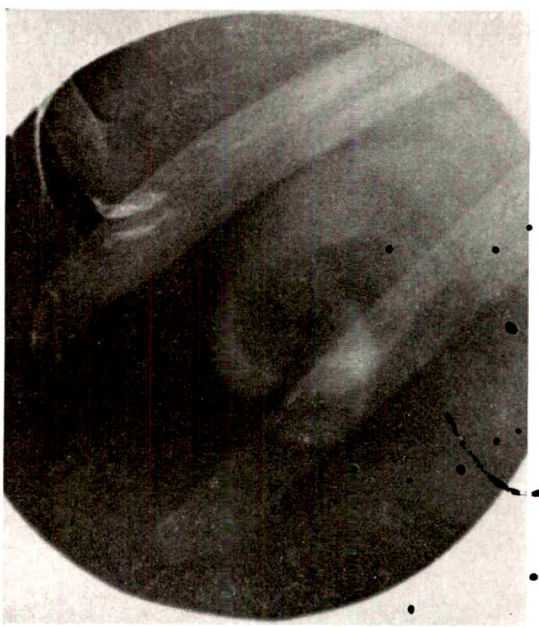


FIG. 10. Medium sized gallbladder of fair concentration. There are vacuolated areas in the region of fundus.

radical has added to the difficulties in the use of these preparations.

For the past year, I have administered a new drug with which these major objections have been largely overcome. This drug is beta-(3,5-diiodo-4-hydroxyphenyl) alpha phenol propionic acid, known under the trade name of priodax.* This new substance has no phenolphthalein radical in its composition and is totally unrelated to it. Priodax contains 52 per cent iodine, in firm combination, which absorbs roentgen rays strongly, but with no danger of dissociation, and therefore no possibility of iodism. It is a light, cream-colored powder and is practically tasteless. This new contrast medium is administered in tablets which may be swallowed or dissolved in water.

Priodax is easy to take and is well tolerated. With the elimination of the phenolphthalein radical, certain disadvantages of the old gallbladder dyes have been removed. One of the major advantages is the avoidance of any laxative action. The laxative effect of sodium tetraiodophenolphthalein is not only objectionable because of the inconvenience to the patient, but also because of the resultant decreased absorbability of the compound. Animal toxicity studies by Modell[5] showed no deleterious effects on the kidneys or liver. In addition to having no laxative action, priodax causes no severe gastrointestinal reactions. Vomiting rarely occurs, with the result that none of the drug is lost to lower its effectiveness.

In our series of 143 cases, there was 3 per cent of vomiting and negligible intestinal symptoms. It was evident that the absorption of the dye was satisfactory by the great percentage of good fillings and homogeneous shadows (Table 1).

There were no contrast-filled gas shadows obscuring the fundus of the gallbladder. The superimposed contrast-filled

* Made by the Schering Corporation.

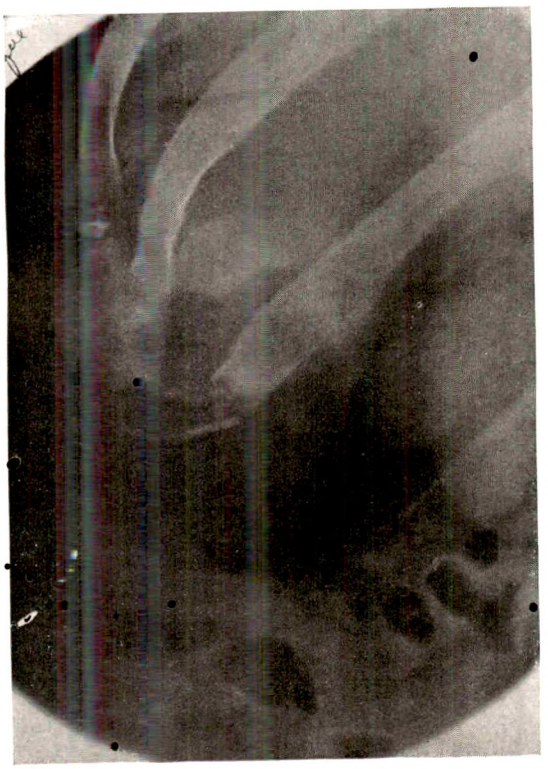


FIG. 11. Oblique view of the same case as Figure 10 shows the vacuolated areas to be within the fundus and are characteristic of calculi.

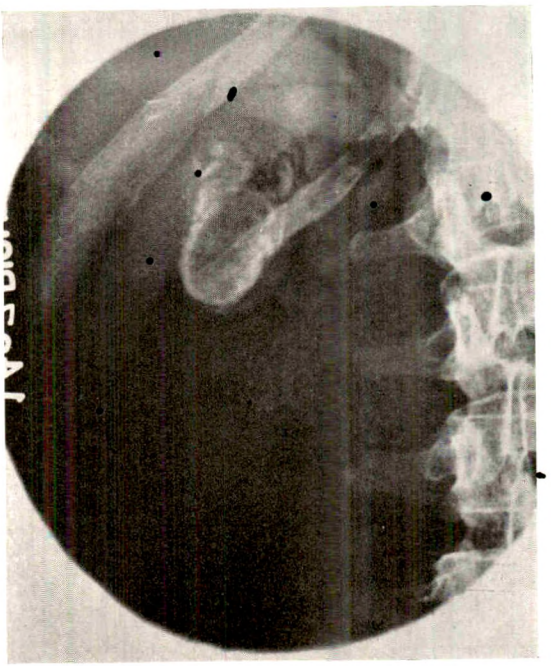


FIG. 12. Numerous vacuolated, nucleated, calcified shadows within the fundus. Operative findings March 18, 1943, cholelithiasis and large stone in cystic duct.

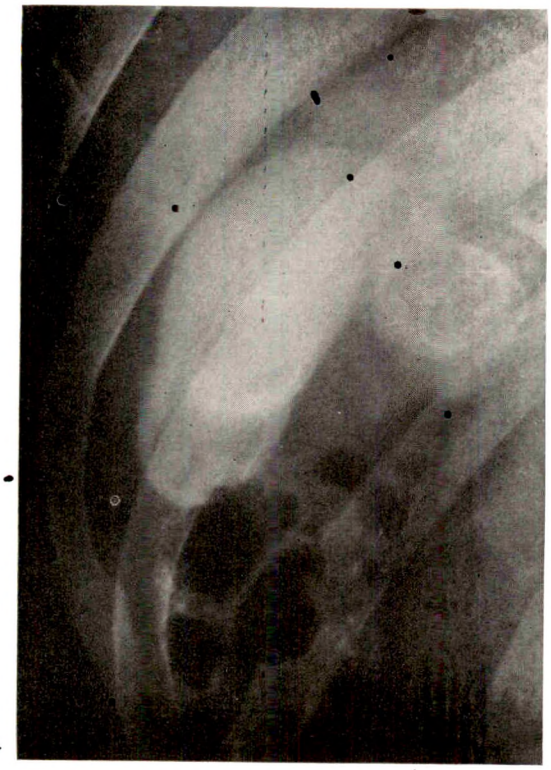


Fig. 13. I.S. Weight, 200 lb. Double priodax. Oblique view shows medium sized gallbladder of good concentration. Vacuolated areas within the fundus which are characteristic of cholelithiasis.

intestinal shadows are seldom seen and the gallbladder can be easily identified.

The fact that priodax is taken orally eliminates many of the disadvantages of intravenous administration. For years, a drug has been sought which would be safe and at the same time effective when given by mouth. From my experience with priodax, it seems apparent that this new substance fulfills these requirements well. The use of a contrast medium intravenously has proved too toxic in many instances. Besides the toxicity, the possibility of producing thrombosis of the vein or necrosis of the perivascular tissues by accidental infiltration has always been a danger with intravenous administration. With priodax, we have a substance which is relatively free from side effects, conveniently and simply administered, and which produces excellent roentgenographic shadows.

TECHNIQUE

(1) The control roentgenogram is taken prior to the ingestion of the contrast medium.

(2) The patient is given a carbohydrate lunch.

(3) Six tablets (0.5 gm. priodax) are dissolved in one-fourth glass of water and one-fourth glass of fruit juice and then swallowed. The tablets may also be swallowed whole at ten minute intervals.

(4) No supper is taken.

(5) No breakfast is taken.

Table I

CHOLECYSTOGRAPHIC STUDIES WITH PRIODAX

Total cases	143	
Females	104	
Age 20–30	12	
31–40	33	
41–50	31	
51–60	14	
61–70	8	
Age not stated	6	
Males	39	
Age 10–20	2	
21–30	6	
31–40	11	
41–50	7	
51–60	12	
61–70	1	
Age not stated	0	
Method of Administration		
Swallowed whole	22	
Dissolved in water with fruit juice added	121	
Degree of Visualization		
Good	101	
Fair	11	
Poor or none	31	
Side Effects		
Nausea	24	7 slight
Diarrhea	33	3 slight
Vomiting	3	
Burning or pain on urination	24	13 slight
males		14
females		10
Griping	29	13 slight 2 epigastric
Headache	2	
Burning in throat	6	
Belching	2	
Bitter taste	2	
"Greasy" taste	1	
Pain in back	1	
Severe reactions	0	
No reactions	9	

(6) The cholecystograms are taken fifteen hours after the oral ingestion and also after a fatty meal.

(7) If there are any gas shadows, an oblique view is always taken to throw the colon away from the fundus. This view is very valuable because gas shadows may be mistaken for calculi and also because small intrinsic calculi may be obscured by the colon. This is time saving and preferable to the use of the pressor agents, as advised by some.

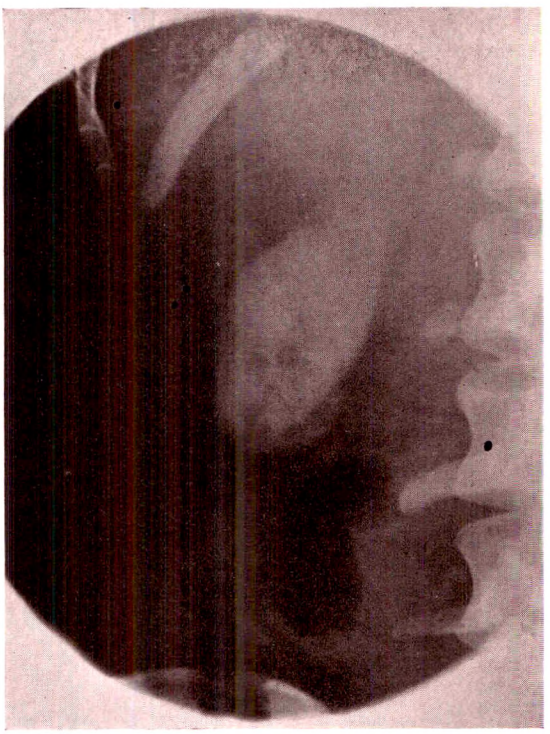


Fig. 14. Medium sized gallbladder of good concentration. Many vacuolated, faceted shadows within the fundus which are characteristic of cholelithiasis.

In our series the concentration of fair and good was over 85 per cent. The assurance of good concentration and certainty of identifying the gallbladder simplify the examination and conserve time. The clear, definite gallbladder shadows produced show the high rate of excretion through the liver. Optimal contrast density occurs in thirteen to fifteen hours. The clarity and distinctness of the gallbladder shadows with the use of priodax permit reliable diagnostic

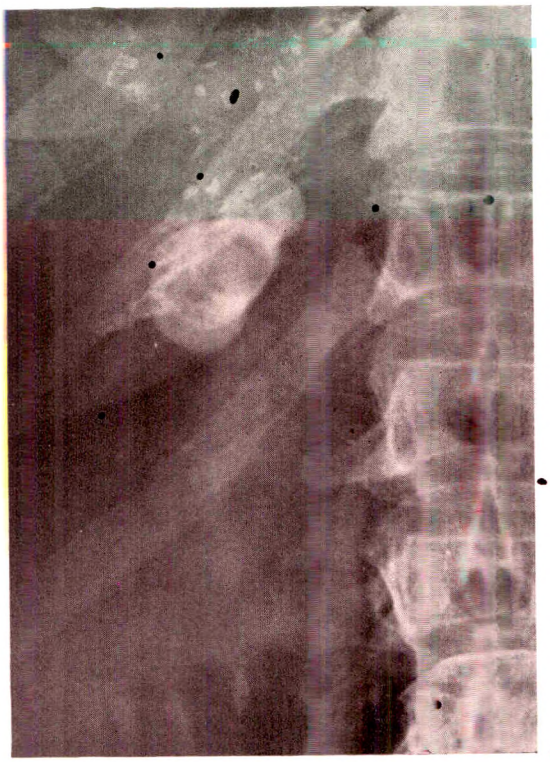


Fig. 15. Medium sized gallbladder of good concentration. Numerous ovoid faceted, vacuolated shadows within the fundus. A constriction in the proximal portion is probably ascribable to pericholecystic adhesions.

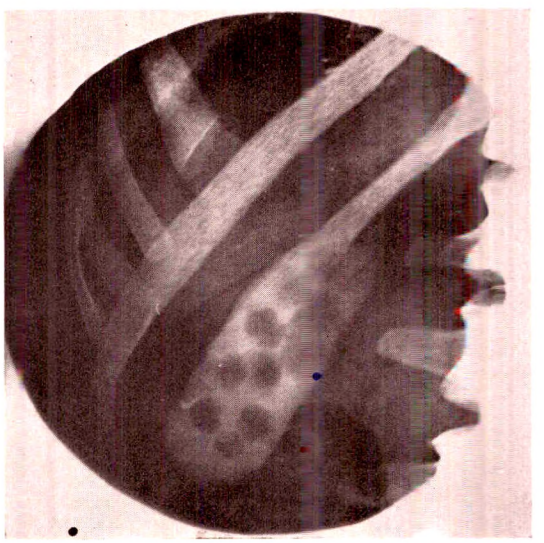


Fig. 16. Numerous sharply defined, vacuolated shadows within the fundus which are characteristic of cholelithiasis. Operative findings, September 16, 1942, confirm cholelithiasis.

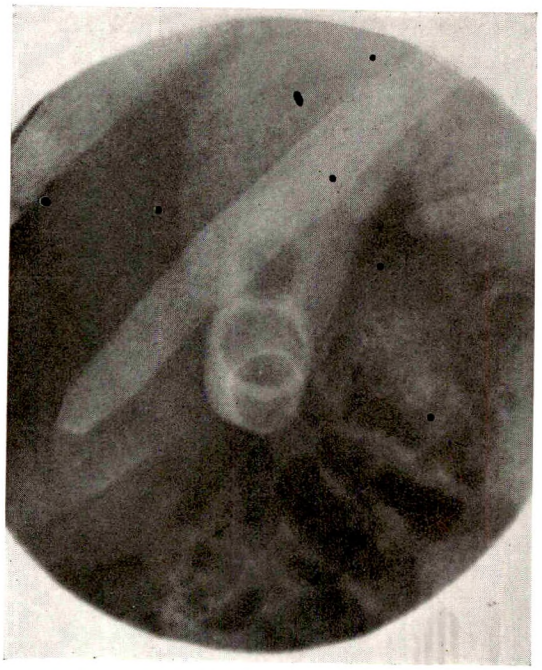


FIG. 17. Medium sized gallbladder of good conc
tration. There are several vacuolated, ovoid ar
filling the fundus. Gas in the colon is thrown av
from the gallbladder shadows. Gas in the hep
flexure is thrown away in this view clearing
fundus. Operative findings: November 27, 1
cholelithiasis.

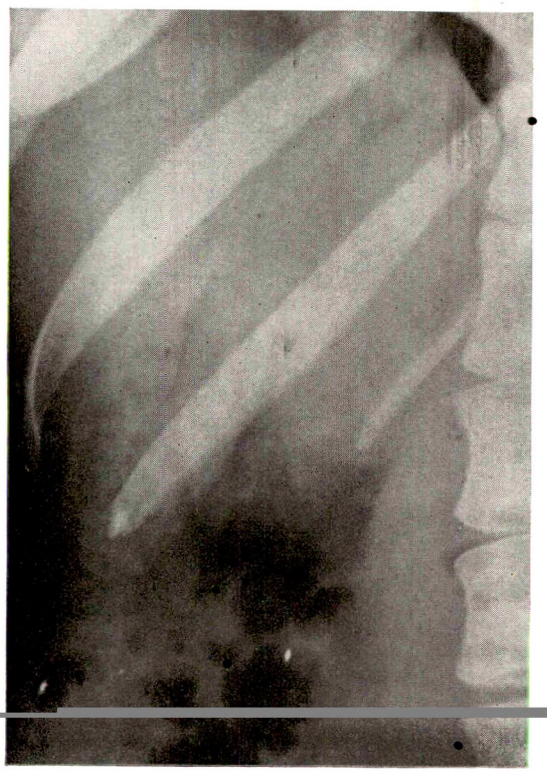


FIG. 18. Medium sized gallbladder with numerous vacuolated areas within the fundus which are characteristic of lithiasis.

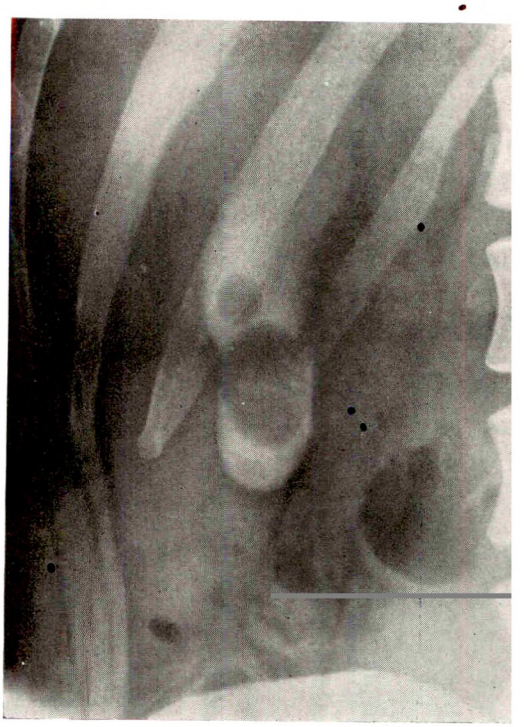


FIG. 19. Medium sized gallbladder with several w
delineated, ovoid, vacuolated areas filling t
fundus. Oblique view clears gas in the hepa
flexure away from the fundus.

differentiation between the visualized no
mal gallbladder and the non-visualiz
pathological gallbladder. At the same tim
in cases of cholelithiasis, the shadows a
light enough to permit good contrast wi
gallstones. This is of diagnostic value sin
too dense shadows may lead to errors by t
masking of gallstones of the millet se
type.

SUMMARY

(1) The new contrast medium, prioda
is easy to take.

(2) There is a low incidence of vomitin
diarrhea and abdominal irritative rea
tions.

(3) There is a concentration of the contrast medium in a high percentage of cases.

(4) There are no obscuring contrast-filled intestinal shadows.

(5) No repeat examination is necessary.

(6) No alkalies, drugs, pressor agents or paregoric are necessary.

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CALCIFICATIONS OF THE SPLEEN

By EDWIN F. GRAY, M.D.*
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CALCIFICATIONS of the spleen are occasionally encountered during roentgenographic examination of the abdomen or during necropsy. The literature contains numerous articles pertaining to this subject, and in view of the controversial nature of the reports of the etiology, this investigation was conducted in an effort to bring forth additional facts.

LITERATURE

The type of calcification most frequently reported is the small, multiple form, sometimes described as the miliary type. The first report was made by Mitchell[37] in 1919. Koppenstein[31] (1927) presented 8 case reports and maintained that they were due to calcified venous thrombi (phleboliths). However, he did not have microscopic proof in any of the cases. Courtin and Duken[12] reported 3 pediatric cases, thought to be of tuberculous origin, as there was demonstrable tuberculosis in other parts of the body. In one instance, they noted progressive calcium deposition in the spleen on a series of roentgenograms. Courtin and Duken stated that the cases reported by Koppenstein were similar to theirs and, as tuberculosis was present in other parts of the body, they should be considered tuberculous. Hellgren[23] observed the development of small calcified areas in the spleen of a child. Sweany[62] stated that 24.6 per cent of the patients who died of tuberculosis in the Chicago Municipal Tuberculosis Sanatorium had roentgenographic evidence of calcifications in the spleen. Sweany and Martinsen[60] reported 2 cases of calcified tuberculosis of the spleen confirmed by necropsy. In 1 case Spitz[56] based the diagnosis on the associated finding of renal tuberculosis and calcified pulmonary tuberculosis. Bársony and Schütz[3] reported phleboliths in 4 per cent of 180 spleens studied by roentgen examination and confirmed by microscopy. Berman[4] reported 18 cases, encountered during routine roentgenography of the abdomen. He considered the etiological factors of these cases to be: phleboliths, 11; questionable tuberculosis, 4; and doubtful 3. The 4 cases of questionable etiology were so considered because tuberculous lesions were found in other parts of the body. During routine roentgenography of 350 patients, Rudisill[47] discovered 4 instances of miliary splenic calcifications which he believed to be phleboliths, but offered no pathological proof. Henke and Lubarsch[24] discussed calcification of venous thrombi and presented as an illustration a case with many nodules. Moorman[39] reported 5 cases, 1 of which included necropsy findings. In 1 instance, fifty-six calcified nodules were found, while in another, there was associated calcification of the splenic artery. Dickson[13] reported 3 cases diagnosed as tuberculosis. In a pathological and roentgenological study of the evolution of calcified lesions (principally of the spleen) Sweany[58,63] conducted a study on a series of 20 tuberculous cases consisting of 16 adults and 4 children. Of these lesions, 16 had corresponding ages of lesions in the lungs, lymph nodes and spleen; 1 in the mesentery and spleen; 1 in the lungs, lymph nodes, liver and spleen; and in 2 there was no agreement of ages of the respective lesions. Bachman[1] reported 3 instances of calcified phleboliths but after further study[2] they were proved not to be phleboliths. Reichle and Work[46] encountered 71 spleens with calcified lesions during postmortem studies. Twenty guinea pigs were inoculated with material from the lesions in 14 cases. Positive "takes" were obtained in 4 animals from 3 of the cases.

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Precautions had been taken to prevent contamination of the material, and no case was included if there was associated active miliary tuberculosis. Von Orelli,[41] Harrison,[20] Hektoen and Riesman,[22] Brown and Webb,[7] Camp,[11] Teschendorf,[64] and Ruhnau[48] also made reports on the miliary type of calcification. Wydler[68] in 1911 published the results of an extensive investigation, having demonstrated stones in ectatic vessels of the spleen. Jablons[28] reported a proved case in which marble-sized calculi were found in ectatic, hilar branches of the splenic vein. Möschl[38] reported 2 cases which on roentgenographic examination had multiple calcified areas. After reviewing the patients' history and the pathological findings, they were interpreted as inflammatory disease with secondary necrosis followed by calcium deposition. Meyer[36] reported a case having multiple calcified lesions as one of malarial thrombosis after finding concretions in the larger trabecular veins. Bignami and Secomandi[5] reported a case interpreted as being calcified pentastomiasis.

An entirely separate entity is that of the so-called primary tuberculosis which may be designated as such when that disease process may localize itself in the spleen and not only bring about various lesions, but act as a focus for dissemination of bacilli while the original focus may be healed entirely.[8]

Shands[53] and Bunch[8] reported instances of primary tuberculosis of the spleen with associated calcium deposition in which roentgenographic examination of the abdomen disclosed the presence of numerous calcified areas in the spleen. These nodules, however, were larger than in the foregoing type, measuring about 1 cm. in diameter. Three of the patients were treated by splenectomy and the diagnosis was confirmed. In 1 instance the nodule was reported to be due to an old bovine tuberculous infection.

Calcification of an aneurysm of the splenic artery is usually encountered on the roentgenogram as a circular area of calcium density in the region of the splenic artery. Heatley[21] observed 6 such cases. Tixier, *et al.*[65] reported a case in which two saccular aneurysmal dilatations of the splenic artery were present. Seids and Hauser[52] reported the case of a patient having a palpable mass associated with a 9 cm. calcified area medial to the spleen and interpreted as being a calcified aneurysm of the splenic artery. Israeleski,[27] Säfwenberg,[49] Sperling,[55] Högler,[25] and Lindboe[33] reported instances of calcified aneurysm which were proved at operation or necropsy.

Intracapsular hemorrhage with subsequent calcium deposition was reported by Mount, Mount and Hunter,[40] in which instance a female, aged forty-four, was found by roentgenographic examination to have a large area of calcium density in the splenic region. Her history pointed to an automobile accident three years previously, at which time moderately severe pain was present in the left upper quadrant of the abdomen. By ruling out other etiologic factors, they concluded that, at the time of the injury, intracapsular hemorrhage occurred and was followed by calcium deposition.

Calcified hemangioma or lymphangioma has been described by Ernst, Bowen, Ruggers, Wakeley, Hulten and Cowen. Ponzio[44] describes such a case, diagnosed because of its similar appearance to those previously described. Cole[11] reviewed the literature on cavernous hemangioma of the spleen, none of which were found to have calcified areas.

Calcification of the splenic capsule has been reported by Stott and Cotton-Cornwall[57] in which instance there was extensive calcification of the diaphragmatic surface. There was no evidence to indicate that the plaque was due to splenic inflammation or to direct spread from the thorax. Kröber[32] encountered another case, that of a fifty-five year old East African native who had generalized amyloidosis in addition to a draining sinus in the left upper quadrant of the abdomen. Operation disclosed a large spleen completely ensheathed in a bony capsule, broken in one place which was the origin of the tract. The etiological factor was not determined. Pallase, Santy and

TABLE I

Case	Age	Sex	Weight of Spleen (gm.)	Evidence of Systemic Tuberculosis	Roentgenogram of Spleen (Number of Lesions)
1*	44	M	100	None	12
2*	61	F	160	Healed tuberculosis of the peritoneum (miliary), lungs, mesenteric lymph nodes and spine. Caseous tuberculosis of adrenal	5
3*	65	M	120	Calcified tuberculosis of the liver	20
4	70	F	90	None	Calcification of splenic artery
5	60	F	70	None	2
6*	63	M	200	Calcified nodule in liver, possibly tuberculous	1
7	38	M	80	None	Calcification of splenic artery
8	45	F	220	None	5
9	60	M	210	Caseous pulmonary tuberculosis	6
10*	58	F	285	Healed tuberculosis of hilar lymph nodes	Calcification of capsule 8
11*	53	F	60	Healed apical tuberculosis	1
12*	49	F	140	Calcified tuberculosis of lung	3
13*	50	F	270	Healed apical tuberculosis	1
14*	35	F	260	Healed apical tuberculosis	1
15*	70	M	130	Apical scar	1
16*	2½	F	45	None	2
17*	53	M	280	Apical scar	3
18*	56	M	105	Calcified lesion of liver, possibly tuberculous. No apical scar	Calcification of capsule
19*	67	F	105	Apical scar	11
20*	54	F	260	Apical scar	2
21*	50	F	75	Healed tuberculosis of lung	1
22*	71	M	140	Adhesions of apical pleura	1
23*	50	F	220	Not examined	2
24*	58	F	85	Apical scar	Infarcts with calcification
25*	43	F	200	None	2
26*	21	F	170	No apical scar	2
27*	31	F	180	Tuberculosis of mesenteric lymph nodes	4
28*	37	M	250	None	1
29*	43	M	260	None	2
30*	54	M	200	Apical scar	2
31*	59	M	240	Tuberculosis of lung, spleen and hilar lymph nodes	4
32*	18	F	110	None	2
33*	58	M	340	None	Calcification of capsule
34*	63	F	710	Healed tuberculosis of lung, tuberculosis of tonsil	16
35*	45	F	140	Thickening of apical pleura	6
36*	41	M	440	Healed tuberculosis of lung	6
37*	73	M	200	Healed tuberculosis of lung	2
38*	80	M	260	Tuberculosis of lung, ileum	3

TABLE I—*Continued*

Case	Age	Sex	Weight of Spleen (gm.)	Evidence of Systemic Tuberculosis	Roentgenogram of Spleen (Number of Lesions)
					Calcification of splenic artery
39*	28	F	110	Lungs not examined	3
40*	41	M	170	Healed tuberculosis of lung	1
41*	80	M	60	Healed tuberculosis of lung	9
42*	59	F	110	Apical scar	1
43*	42	M	150	Healed tuberculosis of lung	6
44*	76	M	180	Thickening of apical pleura	8
45*	55	M	140	Healed tuberculosis of lung	5
46*	66	M	100	None	Calcification of capsule
47*	50	M	200	Healed tuberculosis of lung	2
48*	38	F	460	Apical scar	3
49*	48	M	250	None	1
50*	69	M	100	Healed tuberculosis of lung	4
51*	72	F	100	Apical adhesions	1
52*	66	M	280	Healed tuberculosis of lung	4
					Calcification of splenic artery
53*	55	M	180	Apical scar	2
54*	55	M	260	None	2
55*	33	M	220	None	4
56	50	F	150	Tuberculosis of the liver (miliary) and of the stomach, lung, gastric and hepatic lymph nodes	2
57*	72	M	300	Tuberculosis of spleen (miliary) and peritoneum	2
					Calcification of capsule
58*	57	M	320	None	3
59*	35	F	120	None	4
60*	50	M	240	Healed tuberculosis of lung and bronchial lymph nodes	60
61*	48	M	150	None	13
62*	71	F	170	None	4
63*	74	M	160	Tuberculosis of adrenal	12

* Examination included only one-half of the spleen.

Chanaleilles[42] reported an instance of painful perisplenitis with calcification, the symptoms of which were relieved by splenectomy.

Goinard[29] recorded the case of a male, aged fifty-seven, who had jaundice and splenomegaly. Four and one-half years after the onset of symptoms a roentgenogram of the abdomen dislosed calcified areas in the spleen, at which time there was eosinophilia and a positive intradermal reaction indicating the presence of a hydatid cyst. The diagnosis was confirmed by operation.

Three instances of calcium deposition in infarcts were recorded by Kadrnka and Babaiantz.[29] Their roentgenological diagnostic criteria are: (*a*) single or double intrasplenic calcareous foci having a triangular or oval form; (*b*) triangular shadows with apex toward the center and base toward the splenic capsule; (*c*) generally homogeneous calcification, either porous or soft, and (*d*) contours which are sharply

demarcated, but which may show small irregularities indicative of islands of absorption.

Calcified non-parasitic cysts of the spleen are quite rare. Fowler[17] reviewed the literature and mentioned cases reported by Kohnstamm, Piccinilli, Pean and Sphillman. Földes,[16] Gatersleven,[18] and Pool and Stillman[45] reported cases diagnosed roentgenologically. Calcification in the wall of a cyst has been reported by Scotson[51] in which instance a layer of calcium was found to measure $\frac{1}{32}$ inch in thickness. An instance is reported[66] in which roentgen studies disclosed a calcified mass the "size of an apple" in the splenic region which, after removal, was found to contain yellowish fluid and cholesterol plates. Shawan[54] reported an instance of an eighteen year old female who had a huge solitary cyst of the spleen with calcium deposits in its wall. Mattei[35] reported a case with multiple cysts having calcified walls. Bachman[1] presented a case of calcified cyst of the spleen, confirmed by necropsy.

Echinococcus cysts at times have calcium deposits in their walls in which case their roentgenographic appearance is that of a segmented or complete ring-like area of calcium density. Cases with roentgenograms have been reported by Boyd,[6] Downs,[14] Druckmann,[15] Holl,[26] and Calm.[9]

Kaufmann[30] stated that abscesses may calcify and that the *Pentastoma*, a parasitic anthropod, may form calcified nodules the size of a pea, which have been mistaken for phleboliths. Berman[4] cited such conditions as echinococcus cysts and bone forming metastases to be etiologic factors. Henke and Lubarsch[24] stated that trabecular calcification and bone formation occur and that calcium may be deposited in gummatous lesions.

MATERIAL AND METHODS

For this study, a group of 111 spleens from unselected necropsies have been subjected to roentgenographic examination. In most cases only one-half of the spleen was examined. Of this number, 63 (57 per cent) have evidence of calcification, from which the following classification has been made: multiple calcifications of the so-called miliary type, 58; calcification within the capsule, 5; calcification of the splenic artery, 5; and calcification within an infarct, 1. In some instances, lesions of more than one type have been found (Table 1).

Age Incidence. The ages range from two and a half to eighty years and may be classified by decades as follows: first decade, 1; third, 2; fourth, 8; fifth, 12; sixth, 19; seventh, 10; eighth, 8; and ninth, 2.

Sex Incidence. In this series the sex incidence is equal—31 male and 32 female.

Weights of Spleens. The relative occurrence of calcification according to the size of the organ is: 50–100 gm., 8; 100–200 gm., 26; 200–300 gm., 22; 300–400 gm., 3; 400–500 gm., 2; and 600–700 gm., 1.

Evidence of Systemic Tuberculosis. Eight cases have active systemic tuberculosis, while many others disclose evidence of apical scars of thickening of the apical pleura, which are possibly due to healed tuberculosis.

In 2 instances, there are calcified lesions in the liver which were interpreted by the pathologist to be calcified tubercles. Several instances of miliary tuberculosis are present.

Pulmonary infection is the most common site of systemic tuberculosis, while other sites of involvement are: hilar lymph nodes, mesenteric lymph nodes, adrenal, spleen, liver, stomach, ileum, peritoneum and vertebral column.

Multiple Calcifications. Roentgenographic examination shows the calcified areas to be distributed throughout the organ. In number, the lesions vary from one to sixty, but the majority of cases have less than ten. The diameter of the largest measures 8.25 mm. and that of the smallest is a fraction of 1 mm. In each case, one finds the size quite variable and not infrequently several lesions are present in a conglomerate mass. The most frequent configurations are ring like, oval or semilunar. Greater variation in shape is noted in specimens having the

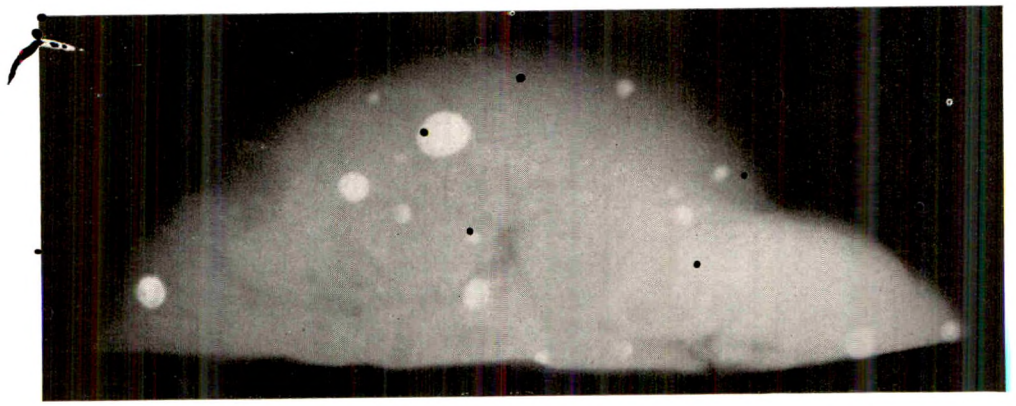


Fig. 1*A*. Roentgenogram of one-half of the spleen showing numerous calcified areas. Note the marked variation in size and shape of the lesions (see Case III).

largest number of calcium deposits (Fig. 1).

On gross examination, the lesions are found in all parts of the splenic pulp and in 1 instance a single nodule is found to be in a splenic fissure outside the capsule. The lesions vary in size from less than 1 mm. to almost 1 cm. The external appearance is that of a translucent, hyalinized mass which consists of a peripheral capsule and a central core. The diameter of the lesion is usually extremely large when compared to the size of the vessels of that vicinity. The capsule is soft and gelatinous and seldom contains calcium. Numerous trabecular and vascular attachments are present on the periphery but no occluded vessels are present. The capsule may be separated from the core and the consistency of the latter varies directly with the amount of calcium deposition. In the presence of little calcium, one finds the lesions to be gelatinous; however, with large amounts of calcium they are extremely hard. In the latter instance, the core frequently has a lamellated structure, consisting of yellowish glistening material which breaks with difficulty.

On microscopic examination, the peripheral portion of the lesion is actually a capsule of concentrically arranged hyalinized fibers. Interspersed with these fibers, one finds elastic tissue, either as loose fibers at the periphery or as one or two delicate layers which completely encircle the capsule. Occasionally the elastic tissue fibers form large masses. In no instance was there extensive calcification, although at times a water-marked appearance was noted as calcium deposits extended from the core. No smooth muscle fibers could be identified. The connective tissue fibers at times appear to be continuous with the trabeculae. No iron pigment is found. Occasionally one finds small areas of bone formation. In only one instance is there small round cell infiltration. Vessels from the trabeculae and pulp, when examined by serial sections, are frequently found to pass into the capsule, but no thrombosed vessels are associated with the calcified lesions. A distinct line of demarcation is usually seen between the capsule and core.

The center of the lesion may be considered as a core. The diameter of this core varies according to the size of the lesion, while the capsule maintains a somewhat uniform width in most instances. The diameter of the core is rather large in comparison with the width of the capsule. The shape varies in each instance, but the general contour is oval or round. The core may be divided into three types in accordance with the manner in which calcium deposition has taken place, as follows: (1) the entire lesion has a hyalinized appearance in which a small amount of calcium is deposited and one finds little to distinguish the core from the capsule; (2) the core is a large, more or less solid mass of calcium sur-

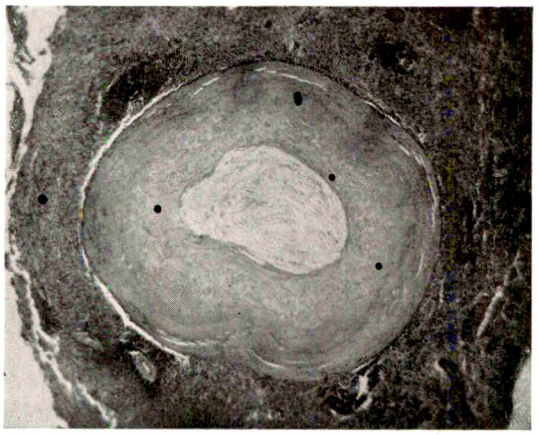


FIG. 1*B*. Photomicrograph (low power) of a single nodule in the spleen. The core contains many cholesterol clefts about which there are calcium deposits. There is no evidence of blood vessel wall or thrombus formation. The capsule has a hyalinized appearance.

rounded by a layer of loose connective tissue which separates the core and capsule; (3) the core is composed principally of loose connective tissue and cholesterol crystal clefts, the center of the core being essentially free of calcium, but peripherally one finds large calcium deposits which extend to and, at times, involve the capsule.

The types described above are not to be considered separate entities, as transitional

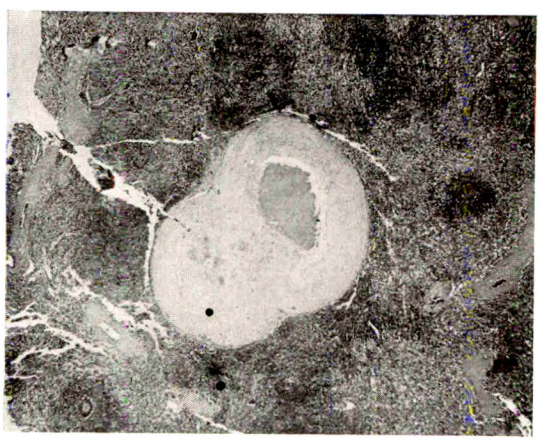


FIG. 1*C*. Photomicrograph (low power) of a double nodule in the spleen, the periphery of which contains numerous small round cells. The lesions have separate capsules which are attached in their periphery.

phases are noted. Occasionally one notes the presence of fibrin, polymorphonuclear leukocytes and lymphocytes, indicative of an inflammatory reaction. No tubercle bacilli were found. Hemosiderin is present in varying amounts, though elastic tissue fibers are not found. Serial sections frequently disclose bone formation, usually as multiple small areas. Bone marrow elements may be included. The calcium deposits appear either as granules or lamellated concretions, with the granular deposits being the most frequent type. The lamellated concretions varied considerably

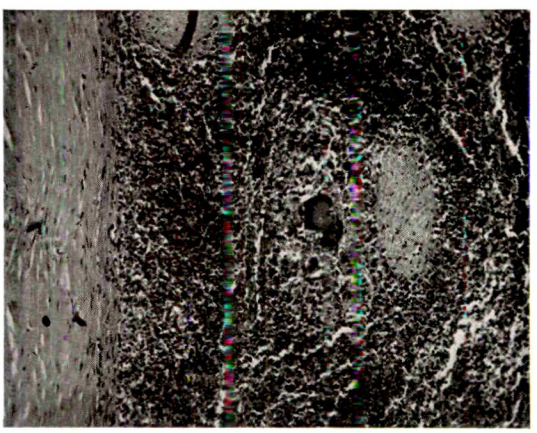


FIG. 1*D*. Photomicrograph (× 110) showing a miliary tubercle immediately beyond the capsule of a calcified nodule.

in size and in no instance was this type found to the exclusion of calcium granules.

The splenic pulp about the lesions is not distorted to any great extent. As mentioned previously, the trabeculae are in close relationship to the capsule. In many instances there are unusually large amounts of hemosiderin about the lesion. Serial sections reveal no blood vessels that end within the wall of the calcified mass to indicate that thrombosis with occlusion of a vessel has taken place. Occasionally a vessel is distorted by the external pressure of a calcified area. The splenic veins are found to follow a rather straight course and in none is there tortuosity. Langhans' giant cells and tubercles are found in several cases, each of

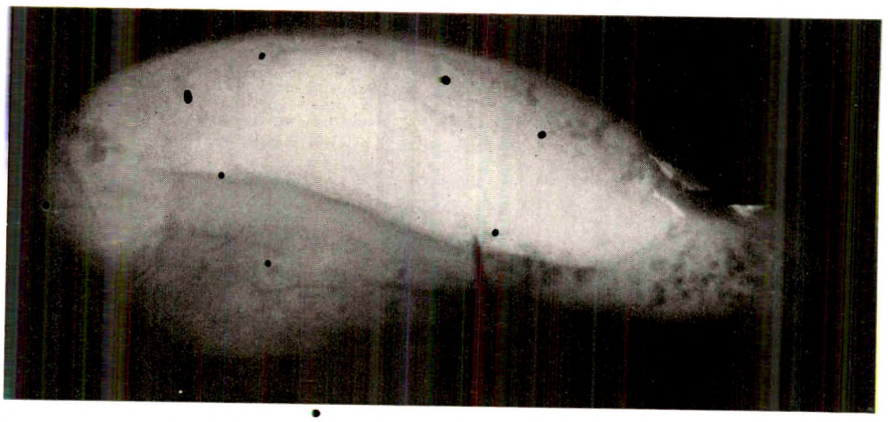


FIG. 2A. Roentgenogram of the spleen, showing mottled calcium deposits within the capsule.

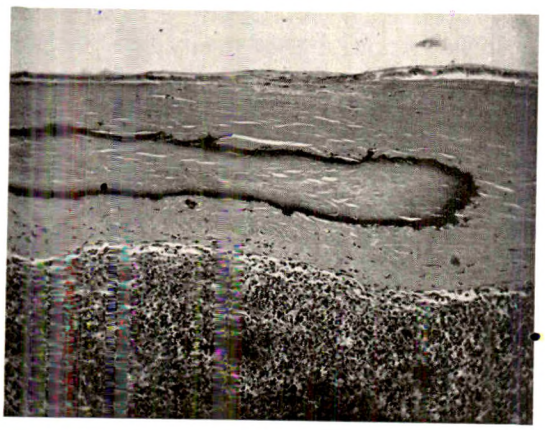


FIG. 2B. Photomicrograph (× 110) showing thickening and hyalinization of the splenic capsule in which there is a large calcium deposit. The adjacent splenic structure is normal.

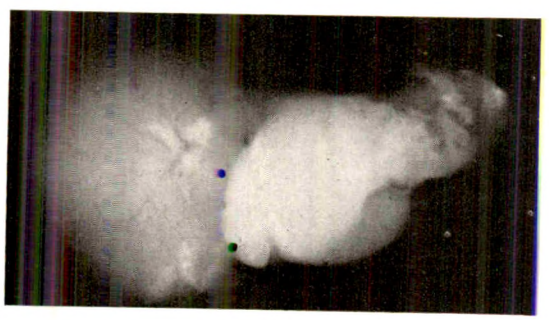


FIG. 3A. Roentgenogram of the spleen, showing mottled areas of calcium density having a triangular shape, the apex of which is directed toward the center of the organ.

Microscopic examination reveals considerable thickening of the capsule, the periphery of which is normal. In the deeper portion there is an unusually large amount of loose fibrous connective tissue. The tissue is relatively avascular and only a few small lymphocytes are present. Granular deposits of calcium are seen in this layer. No bone formation is noted (Fig. 2).

Calcification within an Infarct. In only 1 instance has there been calcium deposition within an infarct. The roentgenogram discloses four triangular areas in which there are deposits of calcium which have a granular appearance, the largest measuring 0.9



FIG. 3B. Photomicrograph (× 40) showing extensive fibrosis about an occluded artery. Numerous calcium deposits and large amounts of hemosiderin are present.

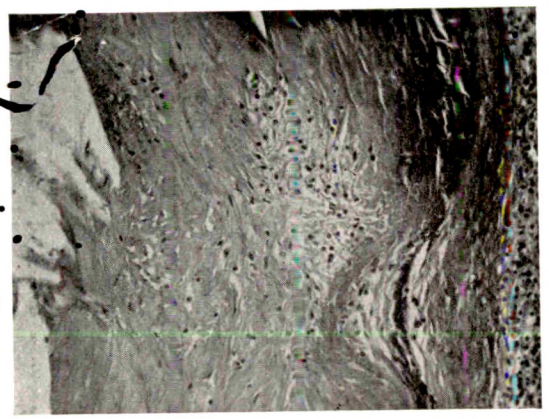


Fig. 1*E*. Photomicrograph (× 230) of a portion of a nodule with many cholesterol clefts and calcium concretions in the center. Numerous lymphocytes are present in the capsule. A thin layer of elastic tissue encircles a portion of the lesion.

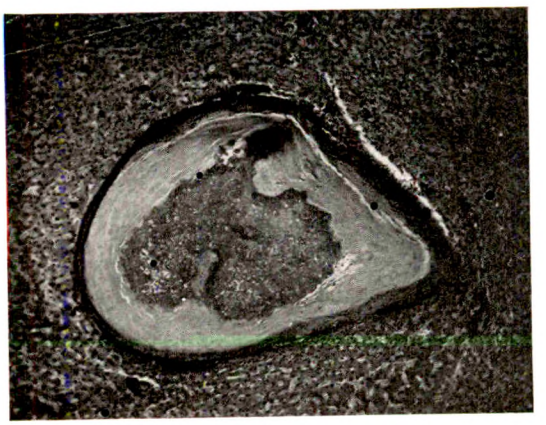


Fig. 1*G*. Photomicrograph (× 110) of a nodule in the liver, consisting of a core and capsule. Large calcium deposits are present in the center. This lesion has the same structure as those previously described in the spleen.

which has active tuberculosis in other organs.

In 2 instances guinea pigs were inoculated with a suspension of twenty-five and thirty fresh, crushed calcified nodules, respectively. At autopsy eight weeks later no tuberculosis was found.

Calcification within the Capsule. Roentgenograms disclosed 5 instances of calcium deposition within the capsule. The calcification is seen as a thin layer composed of granular deposits of calcium density distributed in a linear manner. In 1 case, the deposit included an area 0.5 by 15 cm., while in another it measured 2.5 by 3.5 cm. The areas were smaller in the 4 remaining cases.

Grossly, one finds the capsule to be free of adhesions, except in 1 instance in which there were dense fibrous adhesions to the diaphragm. In all cases there is thickening of the capsule and on section, one encounters calcium deposits. One case shows hyalinization with depression of a 2.5 by 3.5 cm. area of the capsule with calcium deposits limited to this region.

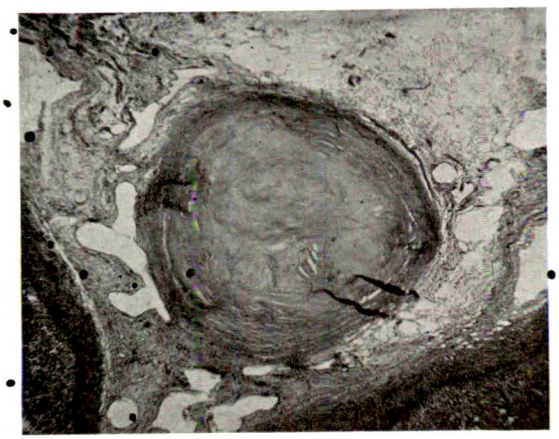


Fig. 1*F*. Photomicrograph (× 110) of a single nodule which lies entirely outside of the splenic capsule.

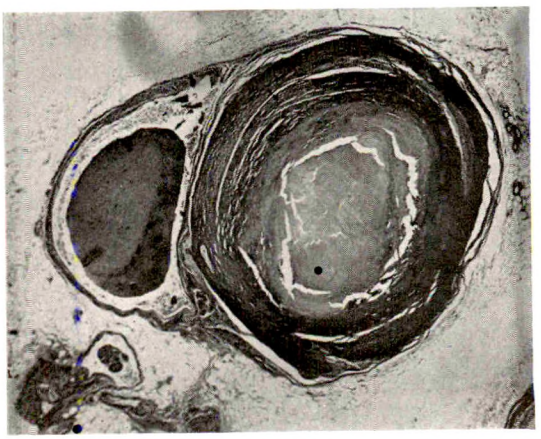


Fig. 1*H*. Photomicrograph (low power) of a cross section of a pelvic vein showing a calcified thrombus and vein wall. Large amounts of hemosiderin are present in the center.

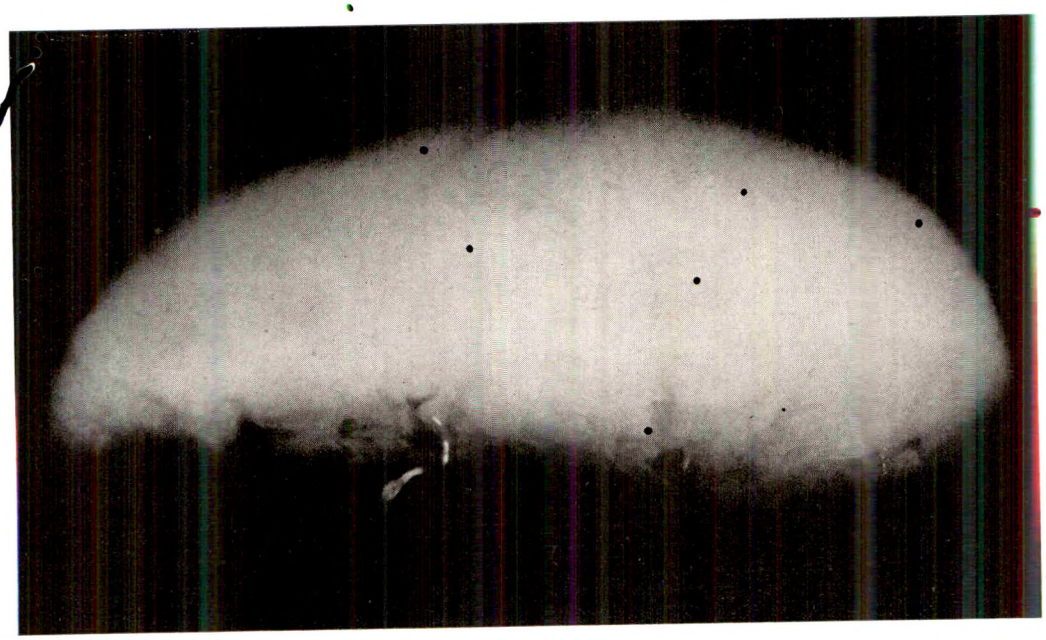


Fig. 4*A*. Roentgenogram of a portion of the spleen, showing calcium plaques within the splenic artery in the region of the hilum.

by 1.8 cm. In each instance the apex is directed toward the center and the base toward the periphery of the spleen (Fig. 3).

The external appearance of the spleen is normal. However, on sectioning, one encounters triangular areas of increased resistance in which calcium deposits are present. The cut surface is slightly darker than the surrounding tissue and has a mottled appearance.

Microscopic examination shows the capsule to be slightly thicker than normal. Immediately beneath the capsule, one sees a sharply demarcated area in which the splenic pulp is replaced by moderately dense fibrous tissue. A small number of small round cells are present, as well as numerous capillaries and small blood vessels. There is a large amount of yellowish-brown refractive pigment. Near the inner margin of the lesion one finds several occluded blood vessels. Diffuse granular deposits of calcium are noted, but there is no evidence of cancellous bone formation.

Calcification of the Splenic Artery. The roentgenographic findings are the same as those of calcification in other vessels, i.e., circular or mottled shadows of calcium density in the course of the splenic artery. These deposits may occur in the splenic artery or in its branches within the splenic pulp (Fig. 4). The calcification may involve only a few millimeters or the entire length of the vessel. However, in this group all calcifications are in the hilum or in vessels

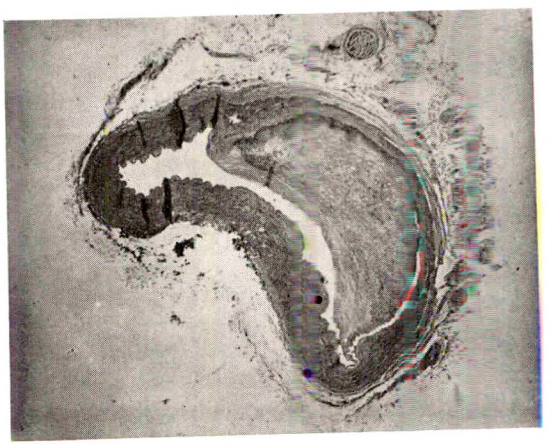


Fig. 4*B*. Photomicrograph (low power) of a section of the splenic artery, showing a large area of intimal thickening in which calcium deposition has occurred. The lumen is considerably narrowed.

within the splenic pulp, but near the hilum.

On gross examination the diameter of the vessel is found to be relatively small when compared to the normal. Microscopic examination discloses marked intimal thickening in the form of a large plaque that is composed chiefly of collagen. In the deeper part, this has undergone degeneration; calcium and cholesterol clefts are found in the degenerated area. In one part there is slight mononuclear cell infiltration in the plaque. There is a loss of nuclei in the media and a deposition of calcium in fine granules. The adventitia is normal.

DISCUSSION

It has been demonstrated conclusively that splenic calcifications of the so-called miliary type occur irrespective of the age or sex of the patient. The number of the calcified lesions varies from one to sixty; however, the majority of cases contain less than a dozen. Actually the nodules are more numerous as only one-half of the organ was subjected to examination in most instances. The roentgenographic characteristics of the lesions are explained by the manner in which calcium deposition occurs. Necropsy disclosed evidence of systemic tuberculosis in a small portion of the cases. However, the majority of the cases were found to have apical scars and thickening of the apical pleura, which are possibly the result of healed tuberculosis.

A single extracapsular lesion was found to contain questionable epithelioid cells and Langhans' giant cells, but no evidence of thrombus formation. In such an instance it seems probable that this lesion is a healed tubercle rather than a phlebolith.

Gross and microdissection, as well as microscopic serial sections, were made on these lesions. Similar studies were made on calcified venous thrombi in the pelvic vessels in order to have a control. In no instance is there direct evidence to indicate that the etiology of the splenic calcifications is either tuberculosis or thrombosis. In addition, no occluded veins are present in the adjacent pulp. The diameter of calcified lesions in the spleen is alw much greater than that of the veins in th region. The splenic veins are not tortu In comparison, the pelvic veins are ma edly tortuous and the diameter of phlebolith is rarely more than twice large as that of the diameter of the asso ated vein. The multiplicity of the lesions one point in favor of miliary tubercu sis as the etiologic factor. Disseminat tuberculosis frequently involves all p tions of the organ but it seems unlikely th thrombi would occur in such a manner. the event that a splenic thrombus shou calcify, it would most likely be associat with residual fibrosis and such was n found here. Bone formation, including bo marrow elements, is present in the sple lesions, as well as in the pelvic phlebolith

Hemosiderin is frequently present small amounts but not in such a manner to indicate the presence of a thrombus.

Considerable elastic tissue is present the wall of the lesions. In a few instanc the fibers encircle the lesions. Howeve they do not have the appearance of th elastic tissue fibers which are found in bloo vessels. Neither can the associated hyali ized fibers be identified as a blood vess wall.

Of all the organs of the body the splee has the maximum phagocytic capacity fo tubercle bacilli.[34,43] Knowing that a larg percentage of the cases were found at ne ropsy to have evidence of systemic tube culosis, it is quite possible that miliary tu berculosis of the spleen was present a some period during the life of the patient. As the most retrogressive process in th course of tuberculosis (Sweany,[59] an Sweany, Tillotson and Kozielek,[61]) on would frequently expect healed tuberculo sis of the spleen. The presence of simila lesions in the liver is evidence favorin tuberculosis as the etiologic factor.

Guinea pig inoculation was performed i 2 cases and after eight weeks there was n evidence of tuberculosis. The inability t find acid-fast bacilli in only 2 cases canno be regarded as conclusive. Reichle an

Work[6] obtained positive "takes" in 4 animals of 3 cases.

Each of the 5 instances of calcium deposition within the capsule is associated with hyalinization which occurs as a degenerative change. In 1 instance there were adhesions between the spleen and the diaphragm, but as the calcium deposits are identical to those in which adhesions are absent, it is improbable that perisplenitis is the etiologic factor. This type of lesion is present only in patients of an advanced age.

The only case of calcium within an infarct is recognized by its gross, microscopic and roentgenographic characteristics.

Four cases of calcification of the splenic artery have been encountered. The calcium deposits represent an advanced stage of arteriosclerosis and involve small areas in most instances. Aneurysmal changes have not been encountered. With the exception of 1 patient, aged thirty, all patients were of an advanced age. Other instances of calcifications of the spleen encountered during routine roentgenographic examination of the abdomen are: multiple calcifications, 50; calcification of the splenic artery, 2; and calcification in an aneurysmal dilatation of the splenic artery, 1 (Fig. 5).

Several illustrative case reports follow.

Case 1 (Unit No. 501768). A white female, aged sixty-three, was admitted April 18, 1938, because of repeated attacks of epigastric pain and jaundice of five years' duration. Nausea and vomiting accompanied the pain. Physical examination was negative.

Roentgenographic examination of the abdomen disclosed the presence of gallbladder calculi. The spleen was normal in size. In the region of the splenic artery there were several sharply demarcated, ring-like shadows of calcium density, the largest of which measured 1.5 by 2.5 cm. A diagnosis of aneurysmal dilatation of the splenic artery was made (Fig. 5).

Cholecystectomy was performed on April 27, 1938. At the time of operation, the splenic artery was found to be firm, tortuous and dilated, with evidence of calcification. These findings confirmed the roentgenological interpretation.

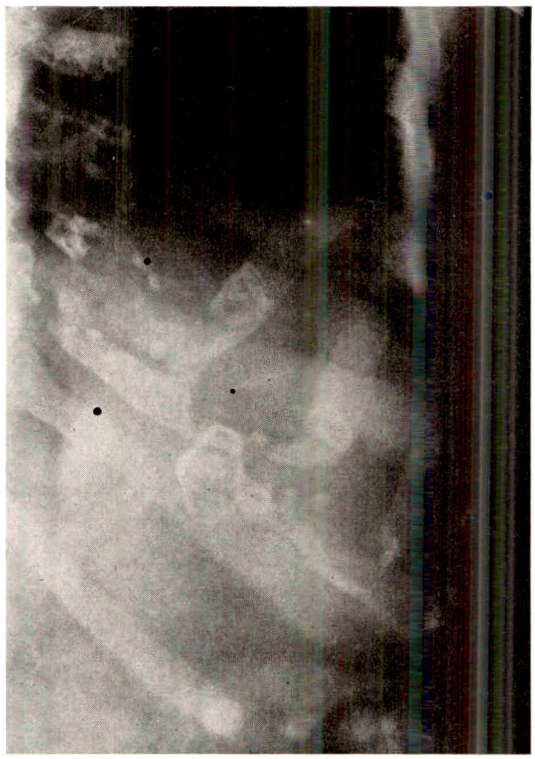


Fig. 5. Case 1. Roentgenogram of the abdomen showing the ring-like shadows in the region of the splenic artery at the hilum. At operation this was found to be a calcified aneurysmal dilatation of the splenic artery.

Comment. A calcified aneurysmal dilatation of the splenic artery was diagnosed by roentgenographic examination and confirmed at operation. No symptoms could be attributed to this lesion.

Case 11 (Unit No. 315675). A white female infant, aged eight months, was admitted October 5, 1931, with a history of pneumonia two months previously, after which the infant failed to gain weight. The illness accompanying the pneumonia persisted for about three weeks. At the time of the acute illness, the patient weighed 15 pounds, while at the time of admission she weighed only 10 pounds. Three days before admission, she suddenly began to refuse all food.

Physical examination showed the patient to be dyspneic, markedly dehydrated and undernourished. The temperature was elevated to 101°F. Evidence of acute otitis media, left, was present. Large, firm lymph nodes were

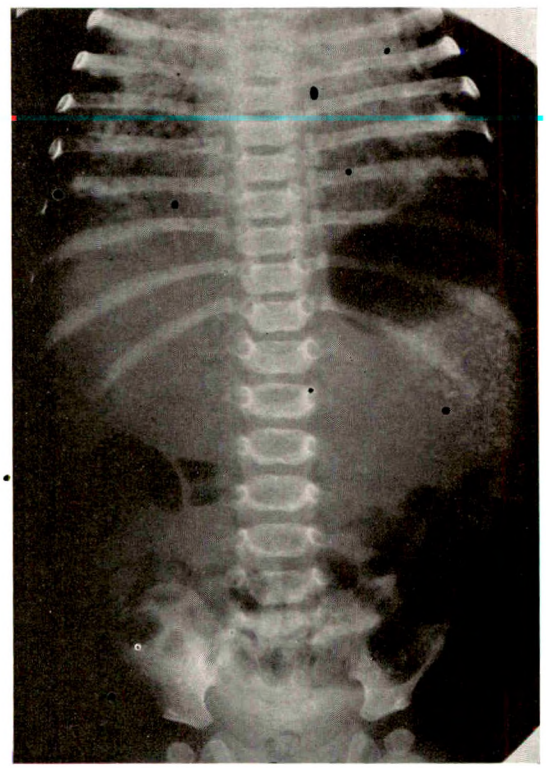


Fig. 6. Case II. Roentgenogram showing calcified areas in the spleen, liver and lymph nodes (mesenteric, inguinal and prevertebral). Disseminated pulmonary tuberculosis is present.

palpable in the cervical and occipital regions. There were questionable signs of pneumonia in the left lower lobe.

Roentgenographic examination disclosed the presence of miliary tuberculosis of the lungs. In the region of the spleen and liver there were many small oval shadows of calcium density, predominantly in the former (Fig. 6). In addition, there were shadows in the mesenteric, cervical, inguinal and prevertebral lymph nodes, suggestive of calcium deposits within tuberculous lesions.

Following admission, the temperature was persistently elevated. The râles persisted and signs of cavitation developed in the right apex. Incision and drainage of a cervical lymph node liberated a large amount of greenish pus, in which tubercle bacilli were demonstrated. The patient became more acutely ill and the temperature rose to 105°F. Death occurred thirteen days following admission. Necropsy was not permitted.

Comment. This case is one of acute miliary tuberculosis, complicated by tuberculous otitis media and mastoiditis. Calcium deposits were in the lymph nodes, liver and spleen, demonstrated by roentgen examination, all of which undoubtedly represent calcium deposition with tubercles.

Case III (Unit No. 456835). A white female, aged sixty-one, was admitted January 30, 1936, because of pain in the lumbar spine. A diagnosis of Addison's disease had been made four years previously. Pott's disease had been present for several years and was accompanied by a pain referred to the anterior aspect of the thighs. Mechanical appliances to her spine relieved the pain temporarily.

Physical examination showed the patient to be quite emaciated. A generalized pigmentation of the skin was present. There was a kyphosis at the level of the third and fourth lumbar vertebrae, without associated tenderness.

Roentgenographic examination of the chest disclosed evidence of tuberculosis of the left upper lung associated with calcium deposition. There was destruction in the third and fourth lumbar vertebrae and the adjacent intervertebral disc, resulting in a kyphosis. Examination of the abdomen revealed calcium deposits in the mesenteric lymph nodes (Fig. 7) and spleen.

Course. Three days after admission, the patient became comatose. The blood sugar at that time was 45.2 mg. per 100 cc. She returned to consciousness after the intravenous administration of cane sugar. One day later the blood pressure fell to 82/46 from 140/80 and her temperature became elevated. Physical examination disclosed no cause for the persistent fever, though diminished breath sounds were noted in the right lower chest posteriorly. The blood pressure remained lower than normal and death occurred February 10, 1936.

Necropsy disclosed the presence of healed tuberculosis of the lungs, retroperitoneal lymph nodes and peritoneum (miliary). A kyphosis was present in the lumbar region due to destruction of the third and fourth lumbar vertebrae. Fibrocaseous tuberculosis of the adrenals was present. Numerous calcified areas were present in the spleen.

SUMMARY

1. The literature on calcifications of the spleen is reviewed.

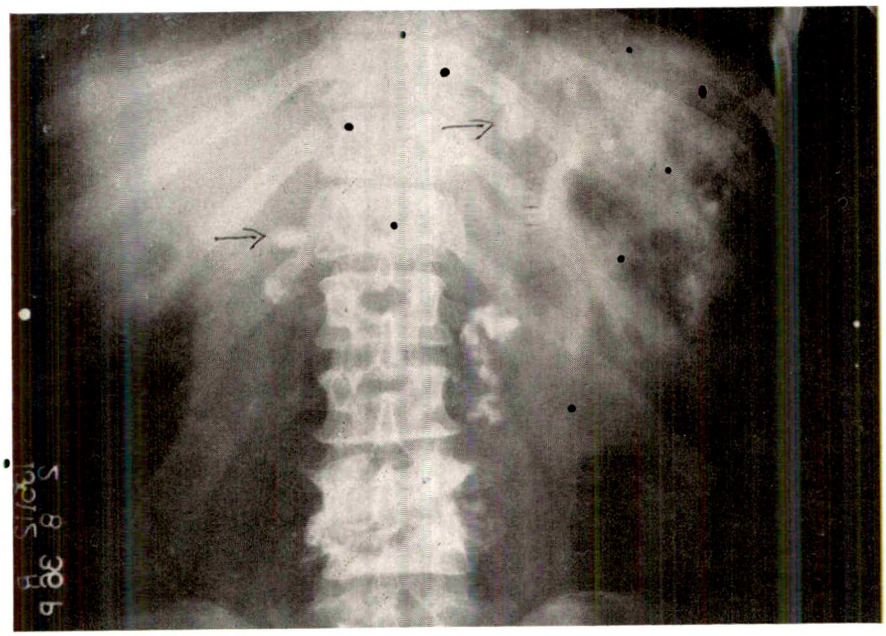


FIG. 7. Case III. Roentgenogram of the abdomen, showing calcified areas in the spleen and mesenteric lymph nodes. Tuberculosis of the third and fourth lumbar vertebrae is associated with destruction of the intervertebral disc and partial collapse of the vertebral bodies. The shadows indicated by arrows are intestinal artefacts.

2. Calcifications of the spleen are frequently encountered during roentgenographic examination of the spleen and at necropsy. Sixty-three cases are reported, an incidence of 57 per cent.

3. The most frequent types of lesions are: miliary calcified lesions, calcification of the splenic artery, calcification of the splenic capsule and calcium deposition within healed infarcts.

4. Miliary calcification may occur at any age, but most commonly after thirty years of age. Calcifications of infarcts, the capsule and splenic artery usually occur in patients of advanced age.

5. The so-called miliary calcifications apparently represent calcified miliary tubercles.

6. Evidence of systemic tuberculosis, active or inactive, is present in most instances.

7. The roentgenologic and pathologic features of the various types of splenic calcifications are discussed.

8. Other instances diagnosed by roentgenographic examination during life are mentioned.

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CONGENITAL ABSENCE OF THE PATELLAE ASSOCIATED WITH ARTHRODYSPLASIA OF THE ELBOWS AND DYSTROPHY OF THE NAILS

A HEREDITARY SYNDROME*

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SINCE the last part of the nineteenth century there have appeared in the literature, from time to time, isolated reports dealing with the unusual association of malformations of the nails, hypoplasia or complete absence of the patellae, and deformities with impaired function of the elbows. In many cases this triad has been inherited, in varying modifications, in several generations of the same family, while in others, one or more of the cardinal features have been absent or altered. This condition is purely inherited and congenital and is apparently due to some developmental defect in the ectodermal and mesodermal layers of the embryo. In 1897 Little[7] reported 42 cases of congenital absence or delayed development of the patella, collected from the literature, to which he added 3 cases of his own. In his article he mentions a family observed by Sedgwick in four generations of which 18 persons had no patellae and no thumb nails. The majority of these cases were females, but there is no mention as to whether every member was affected or whether the two deformities were always associated. Wolf[23] in 1900 reported a family in which the mother showed congenital absence of both patellae and complete absence of both thumb nails. Her two children, a son and a daughter, inherited the same deformities, while her grandchildren were perfectly normal. In 1903 Most[9] called attention to a family in which absence of the thumb nail and extensive atrophy of the remaining nails of the mother were inherited by all 6 sons. One of the 6 is the father of the child under observation. The patient had, in addition, 5 sisters of which 4 were carriers of the nail anomaly. The patient showed, besides the nail deformities, complete absence of the patellae. There were, in addition, severe deformities of both lower extremities. The other members of the family with anonychia were not examined but it is conceivable that some may have shown absence of the patellae.

Firth[4] in 1912 reported the case of a family in which the mother and 3 children, all girls, showed congenital absence of the patellae and deformity of the nails. The mother, aged thirty-two, showed no patellae and deformed nails. The eldest daughter, aged ten, showed, in addition to these deformities, inability to fully extend the arms at the elbow, while the youngest daughter showed the deformities but was unable to fully extend only the left arm at the elbow. The third daughter, aged three and a half, showed a rudimentary patella on the left while the right was present and appeared normal. Twins and 2 siblings in the same family, all dead, were said to have been normal. An addendum to Firth's article mentioned a family reported by Dr. John Thomson, consisting of a father, mother and 9 children. The mother and 5 of the children showed complete absence of both patellae and deformity of all the finger nails and toe nails. The father and 4 other children were quite normal. The mother showed some slight inability to straighten her right elbow completely and she could not freely supinate her right forearm. The family reported by Rubin[14] consists of 3 members—a mother with no thumb nails and patellae of about one-half normal size, a son with no patellae but normal thumb nails and a daughter with no patellae but with thumb nails partly developed.

Trauner and Rieger[19] emphasize the dom-

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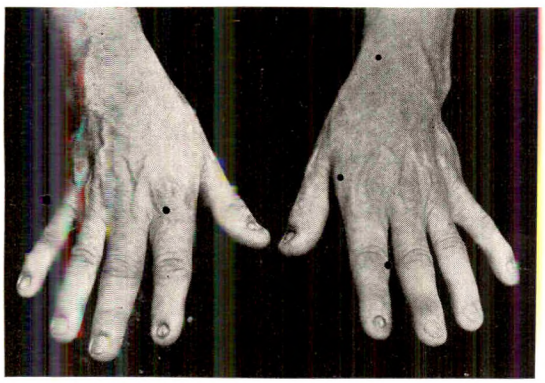


FIG. 1. Hands of patient showing the involvement of the nails of the thumb and index fingers. The nails of the middle and ring fingers are slightly thinner than normal and somewhat flattened. The nails of the little fingers are entirely normal.

luxation, in view of the marked bony overgrowth. On questioning this patient further, it was learned that 2 nieces of his wife and a nephew had rudimentary nails on all fingers, but so far as the patient knew their knees were normal. On his mother's side, a niece and an uncle also had rudimentary nails on all fingers but nothing was known about their knees. The patient died of a cancer of the pancreas and Venable[21] performed an anatomical dissection of the right knee joint which is completely described in a recent report.

Although there has been some variation in the nature and degree of the congenital defects which have been described by the above authors, the localization of the abnormalities to the nails, particularly of the thumbs, to the patellae and to the elbows has been fairly striking. Characteristically, the disorder of the nails has varied from total absence of the nail to one that is only a little thinner than normal, always affecting more severely the thumb nail and becoming less marked toward the little finger. The typical dystrophic nail presents a small normal appearing base that is much thinner than normal and that gradually disappears about half way down the nail bed, allowing the fleshy end of the finger to turn backward over the nail (Fig. 1). Montant and Eggermann have emphasized the fact that only the nail of the thumb was affected and in a characteristic manner, namely, a hemiatrophy of the cubital side. In other cases, there has been complete absence of the nail of the thumb, with or without associated affection of the remaining fingers or, very occasionally, the toes.

The deformity of the patella has been characterized by total absence or at least severe hypoplasia of the patella. The knees appear flat when extended, square and angular when flexed (Fig. 2). The hypoplastic patella, when present, was located on the external aspect of the knees, lying rather high. The tubercle of the tibia is markedly prominent, as is also the internal condyle of the femur. There is genu valgum in some cases. The affected elbow shows an unusually prominent internal condyle, with an increased carrying angle (Fig. 3). The

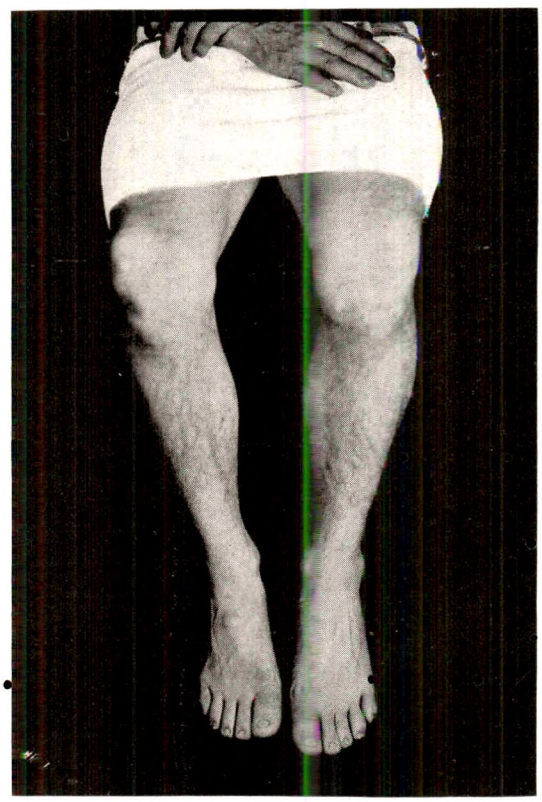


FIG. 2. Knees of patient in flexion showing the square, angular appearance. The nail on the big toe of the right foot shows a suggestive deformity similar to the nails of the thumbs.

inance of luxation of the head of the radius in their report of 6 members in four generations in which they found the following to be closely associated with each other: luxation of the head of the radius caused by hypoplasia or complete absence of the capitate eminence of the humerus, flexion contractures of various fingers, discoloration and incomplete development of the nails of the thumb, index fingers and the first and second toes. In 1 case, there was hypoplasia of the patella. In 1928, Aschner[1] collected from the literature the history of 8 families in which congenital defect or hypoplasia of the patella was inherited through several generations. Among the 8 families, there were 3 in which the bone defect was associated with a hereditary defect of the thumb nails in all the reported affected members of the family.

A complete study is reported by Oesterreicher[11] of a family in which out of 21 members in five generations, 11 were carriers of the defective triad involving the nails, patellae, and elbow joints. There was a defect of the thumb nail in all affected members while 2 showed, in addition, involvement of the other nails. There was a patellar defect in 9 of the affected cases, while in the remaining 2 the information was incomplete in this regard. The anomaly of the elbow was present in 7 of the affected cases, in 2 the information was incomplete, in 1 only the right elbow was involved while in the last case the anomaly was completely absent.

Turner[20] in 1933 reported a study of 2 families in which 35 out of 79 persons were affected by the hereditary triad, namely, dystrophy of the nails, defect or severe hypoplasia of the patella, and deformities of the elbow. In the first family, every member showing dystrophy of the nails suffered also from the other two features, a total of 26 out of 39 persons being affected. In the second family, 27 of 41 persons suffered from dystrophy of the nails while only 9 suffered from the arthrodysplasias. At about the same time, Rutherfurd[15] described a hereditary complex occurring in 5 cases in three generations, characterized by recurrent dislocation of the patella, hereditary knock knee, and aplasia of the nails of the fingers and toes. In 1936 Lester[6] made a supplementary report on the family originally described by Firth in 1912. At this time the family had increased to three generations, of which 7 members of 13 show the deformities. To the relatively constant triad he adds a deformity of the ilium and thickening with convexity of the lateral border of the scapulae. A leaf-shaped discoloration of the iris was observed in a few of the cases.

In 1937, Montant and Eggermann[8] described a hereditary syndrome characterized by hypoplasia of the patellae, malformation of the radius and hemi-atrophy of the nails of the thumbs. These affections were discovered incidentally in an individual who presented herself at the clinic for some trivial unrelated complaint. The genealogical tree was charted when the patient gave the history that several of her ancestors and relatives presented similar malformations. In this way it was possible to determine that in five generations, 10 members of 30 were affected. The authors have emphasized the association of these malformations with the presence of light blue eyes and blond hair, and feel that this is a hereditary affection of a dominant character which is not sex linked. A constant feature in these cases was that only the nail of the thumb was affected and always in the same manner, namely, a hemiatrophy of the cubital side. All cases had a very marked hypoplasia of the patellae, never a complete absence.

Sever[17] reported the case of an elderly man in whom it was discovered, during the course of a routine physical examination, that the patellae were small and located on the external aspect of both knees. It was later observed that the nails on each thumb were rudimentary, while the remaining nails appeared normal. Roentgenograms of the elbow showed various bony exostoses about the joint, but it was not possible to determine the presence of any

forearm cannot be fully extended and supination is limited. Roentgenograms of the elbow show an arthrodysplasia characterized by an elongation and deformity with luxation of the proximal end of the radius. The head of the radius is poorly formed and the superior radio-ulnar articulation is dysplastic (Fig. 4). In other cases there is extensive overgrowth of bone in the region

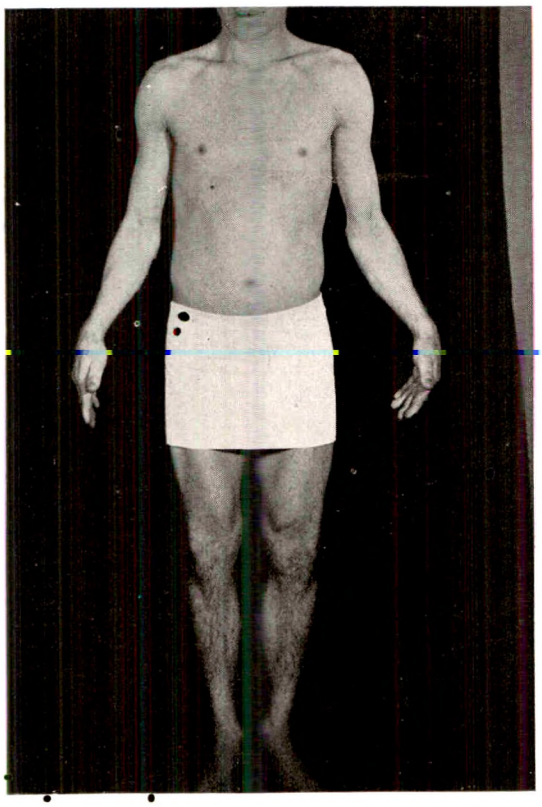


FIG. 3. Photograph of patient standing showing the prominent internal condyles of the lower end of the humeri and the exaggerated carrying angle. The extended knees appear flat with no suggestion of a patella. The hands are held in a pronated position.

of the elbow with secondary arthritic change.

Other less constant and variable features have been stressed by individual authors. Turner[20] called attention to the thick ankles with unusually large malleoli, particularly the internal malleolus. He emphasized the prominence of the acromial ends

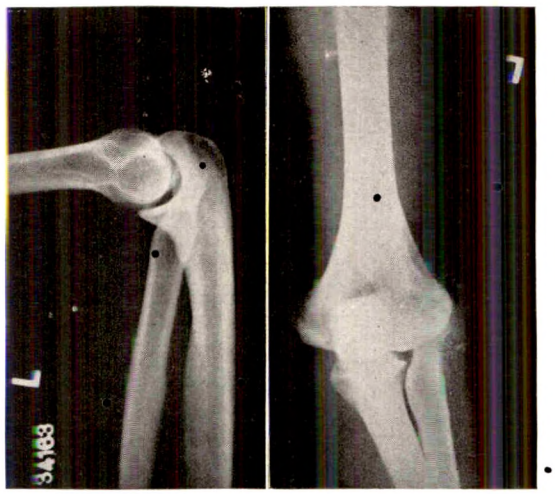


FIG. 4. Roentgenograms of the left elbow showing the deformity of the proximal end of the radius and the dysplasia of the proximal radio-ulnar articulation.

of the clavicle, the undersized scapulae, and the small coracoid and acromial processes. In the hip and pelvis there is said to be an increase in the angle between the neck and shaft of the femur, with an increase in the normal concavity of the external surface of the ilium, giving the crest the appearance of an outward flare in its posterior half. Lester,[6] in addition to the constant triad, called attention to a leaf-shaped discoloration of the iris, an increase in the normal

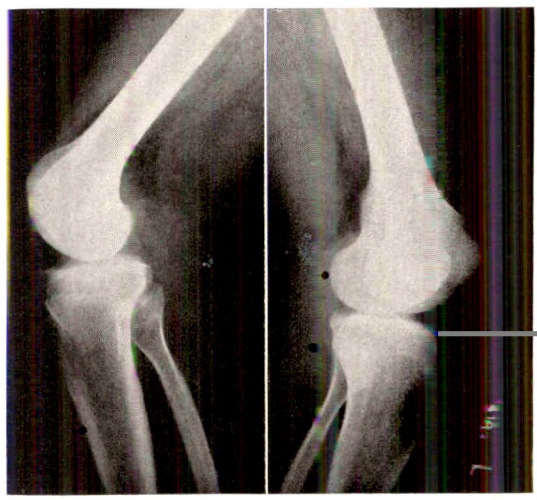


FIG. 5. Lateral roentgenograms of both knees showing complete absence of the patellae.

concavity on the external surface of the ilium so that the crest seems to be increased posteriorly, and to minor changes in the shoulder girdle. Turner, and Montant and Eggermann noted with variable frequency a great laxity of the metacarpophalangeal and interphalangeal joints so that it is sometimes possible to hyperextend the digits of the hand until an angle of 90 degrees is formed with the back of the hand without discomfort to the patient.

knee, the shoulders, wrists, hands, hips and knees. During the course of the examination the deformity of the nails was noted as well as the evidence of total absence of the patellae. Roentgenograms of the knees and the elbow confirmed the clinical suspicion of total absence of the patellae, and established the presence and nature of the arthrodysplasia of the elbow joints (Fig. 4 and 5). The genealogical tree showed that a great-great-grandfather who lived in the eighteenth century is known to have had this affection. The line was apparently negative

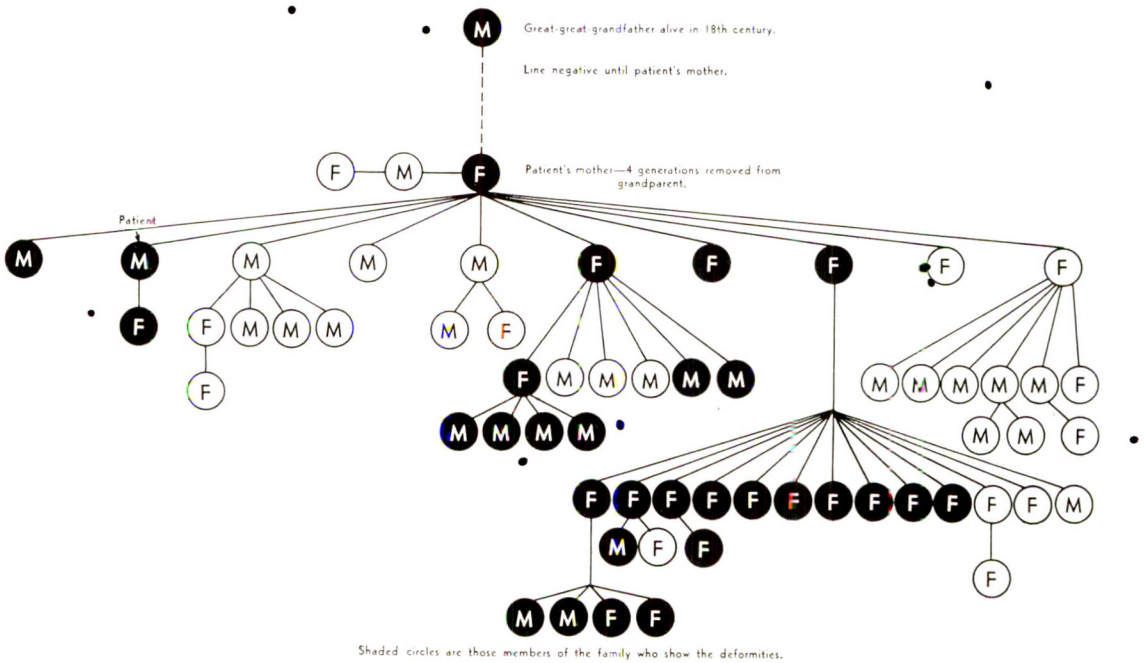


FIG. 6. Genealogical tree of patient showing the involvement of 30 members of the family in four generations. Only the patient was examined personally.

We have had the opportunity to observe a patient who presents the classical triad to which attention is being directed in this paper.

The patient is a male, aged thirty-six, an auto mechanic who was admitted to the hospital because of joint pains following a prolonged siege of boils and lymphangitis, precipitated by his occupation which involved keeping both upper extremities immersed in oil for long periods at a time. The onset of the joint pains and swelling was concomitant with the presence of the boils. The left elbow, which was the site of a large boil, became swollen and painful. This was followed very rapidly by involvement of the right

until the patient's mother who was four generations removed from the grandparent. A total of 30 persons, including the mother, were affected in four generations (Fig. 6).

It is apparent from the literature on this subject that we are dealing with the hereditary transmission of a dominant character, not sex linked, the anomaly being carried at the same time in two different systems, one derived from the mesoderm, the other from the ectoderm. Many genetic theories have been advanced to explain the transmission of these congenital malformations. The most complete and detailed discussion from

the genetic point of view is that of Aschner.[1] She has demonstrated that each of the anomalies can occur independently or in variable combinations with the other two. A defect of the patella can occur independently without involvement of the nails or the elbow joints (Little,[7] Aschner[1]). Hereditary dystrophy of the nails in the absence of skeletal anomalies has been sporadically reported (Tobias,[18] Jacobsen,[5] Pires Da Lima,[12] Ebstein,[3] Murray,[10] Chilton[2]). The hereditary occurrence of luxation of the head of the radius has been described as an isolated anomaly (Servier,[16] Princetau,[13] von Sury[22]). For these reasons, she concluded that there must exist three separate pathologic factors which are closely linked, as the three defects are inherited together with few exceptions. Since the whole syndrome does not occur quite so often as the association of the defect of the nails and the patella, it can be assumed that there is a closer linkage between these two genes in order to explain their frequent occurrence together and their inheritance together as if they were one factor. The genes for the defect of the patella and the thumb nail are not only linked together in the same chromosome, but are even very near neighbors to each other within the same chromosome. The gene for the luxation of the head of the radius is probably in the neighborhood of the other two genes, but not quite as close in the chromosome, as the whole syndrome does not occur quite so often as the association of the defect of the thumb nail and the patella.

Turner[20] postulated that since in neither family in his cases does the arthrodysplasia appear independently of the dystrophy of the nails, although the reverse occurs frequently, the factor for dystrophy of the nails must be present for the factor of the arthrodysplasia to be active. On the other hand, it is possible that both factors are present in every individual showing the dystrophy of the nails, but that a third factor is present also in those who do not show the arthrodysplasia, and the third factor acts as an inhibiting force on the arthrodysplsia or on the interaction of the two. Montant and Eggermann[8] concluded that a single gene is involved in the origin of the different anomalies. This is the concept of polyphenia, which assumes the origin of three different anomalies from a single dominant gene. Trauner and Rieger[19] and Oesterreicher[11] concurred in this assumption.

SUMMARY

An inherited congenital developmental defect involving the ectodermal and mesodermal layers of the embryo is described. The malformations affect, with striking consistency, the patellae, the nails of the hands, particularly of the thumbs, and the elbow joints. This triad is transmitted as a hereditary dominant character and is not sex linked. A case is described showing the typical clinical and roentgenologic appearance, in whose family tree a total of thirty persons in four generations are thus similarly affected.

Acknowledgment is made to Dr. Emanuel Sgobbi, of St. Louis, for authorization to make complete use of the records in this case.

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Vol. 51, No. 3

OSSEOUS ABNORMALITIES OF THE THORACIC CAGE SEEN IN FORTY THOUSAND CONSECUTIVE CHEST PHOTOROENTGENOGRAMS*

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MEDICAL CORPS RESERVE

THE literature on congenital and developmental defects and abnormalities of the thoracic cage is limited. Very little has been written in the radiological journals of this country on the subject, but a number of interesting articles have appeared in foreign publications.[3,4,7,10,11] The roentgenologist meets with these conditions in his daily routine and takes them as a matter of course, reflecting, perhaps, on the infinite variety of biological phenomena.

Of interest chiefly from the standpoint of anatomy, very little was known of the occurrence of thoracic anomalies until the advent of roentgenography. Identification of structural variations by dissection would be a tiring task and this accounts for lack of data on the subject in the anatomical literature and standard texts. The roentgenologist is interested in the abnormalities both from the anatomic and roentgenologic aspect since they at times may simulate pathological conditions as, for example, the mistaking of a bifurcated rib deformity for the wall of a tuberculous cavity. This may seem trite, but it is a fact that the mistake has been made.

While every roentgenologist is familiar with the more common anomalies of the thoracic cage, these are not of such frequent occurrence as to be an every day happening in the average practice. In fact, our statistics on the abnormalities herewith reported would indicate the conditions cited to be rare. Account must also be taken of the fact that this report deals with a segment of the male population of military age, and may therefore be found at variance with statistics accumulated in a study including both sexes.

But up to this time, with the exception of the reviews published by Ashbury,[1] and Ashbury, Whildin and Rogers,[2] no large series of cases giving these data have been reported. Inasmuch as our statistics and those of the authors quoted are substantially in agreement, it is felt that our figures represent a true picture of the occurrence of the various abnormalities which are shown in the accompanying tabulation (Table 1).

COMMENT

The occurrence of cervical ribs in this series, namely once in 600 men, may seem small, but we have held strictly to the following criteria in diagnosing cervical ribs: First, the rib must arise from the seventh cervical vertebral transverse process which is seen to project horizontally from the spine in contradistinction to the transverse process of the first dorsal vertebra which extends diagonally upward from the point of origin (Fig. 1), and second, it must have no connection anteriorly with the manubrium sterni although it may form a synostosis with the first rib (Fig. 4). Many ribs, which at first glance appear to be supernumerary, are found to be rudimentary first ribs when the above criteria are applied. It is of interest that in this series only one of the cervical ribs was found to be jointed (Fig. 2). Vinogradov[12] described 6 cases in which a joint appeared in the mid-portions of cervical ribs. Further, it should be stated that none of the individuals possessing these ribs had any symptoms referable to them at the time of the examination.

Considerable interest has been shown in the appearance of an anomaly of the first rib (Fig. 3) occurring usually in its mid-

* From the Medical Examination and X-Ray Sections, Armed Forces Induction Station, Altoona, Pennsylvania.

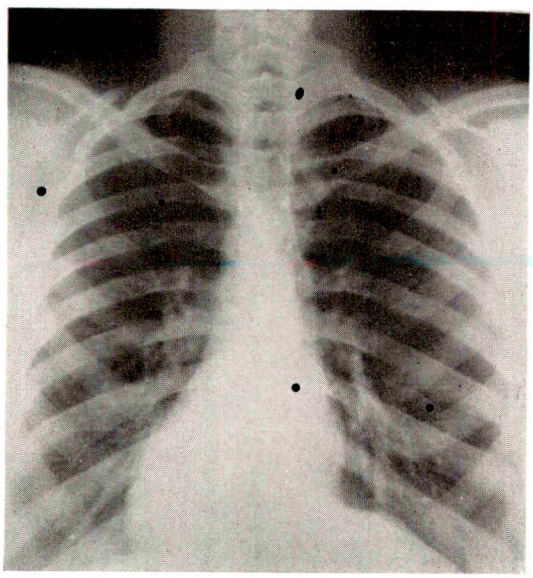


Fig. 1. Bilateral cervical ribs showing origin from horizontal transverse process of seventh cervical vertebra.

portion, and having the appearance of a healed fracture or pseudarthrosis. The accompanying figure shows one case in which the condition was bilateral. When this was first encountered we tried to get a history of injury because of the appearance of callus in a healing fracture, but in all cases the history has been negative. Upon first

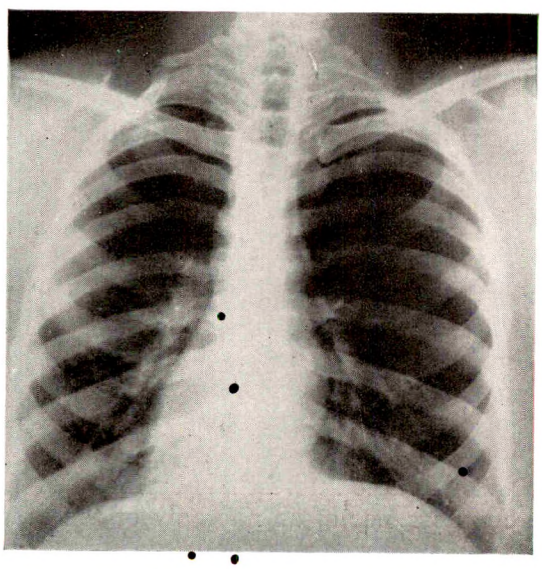


Fig. 2. Bilateral cervical ribs, the left one jointed.

study of a roentgenogram showing this abnormality one is struck by its similarity to a calcified tuberculous process at the apex of the lung. Upon closer examination, and a negative history of trauma, it be-

Table I

Bony Abnormalities of Thoracic Cage in 40,000 Chest Photoroentgenograms

		Right	Left		No. of Cases
Cervical ribs		12	14	26	
		Bilateral		41	
		Bilateral, one jointed		1	68
Anomalous first ribs		12	18	30	
		Bilateral		1	31
Rudimentary ribs	1st ribs	25	42		
	2nd ribs	3	2		
	3rd ribs	2	2		
	4th ribs	0	1		
	5th ribs	0	2		
		30	49		79
Synostosis of ribs	1– 2 ribs	46	31		
	2– 3 ribs	2	2		
	3– 4 ribs	2	1		
	4– 5 ribs	1	3		
	5– 6 ribs	6	2		
	6– 7 ribs	0	2		
	7– 8 ribs	1	1		
	8 –9 ribs	0	1		
	9–10 ribs	2	1		
		60	44		104
	Multiple synostoses				1
	Synostosis rt. cervical and rt. 1st rib				2
	Bilateral synostosis 1st and 2nd ribs				2
Bifid ribs	1st ribs	4	12		
	2nd ribs	3	2		
	3rd ribs	41	23		
	4th ribs	60	33		
	5th ribs	29	18		
	6th ribs	12	5		
	7th ribs	4	4		
		153	97		250
Bilateral bifid ribs	3rd	5			
	4th	2			7
Rib resection (drainage for empyema)					35
Skeletal deformity (scoliosis)					126
Healed fractures of ribs					48
Osteomyelitis of rib					1
Osteochondromatosis					1

comes clear that the lesion is an anomalous pseudarthrosis. Hallermann[5] described a case and drew attention to the appearance suggesting a healed fracture. Pickhan[9] also reported on this condition and conjectured on its possible phylogenetic connec-

tion by showing that similar joints occur in the upper ribs of the European crane.

In this series the most common rudimentary rib was found to be the first. Probably this is the reason that they are sometimes mistaken for cervical ribs although application of the criteria above given for cervical ribs will serve to differentiate the two. None of the individuals we saw had any symptoms from this anomaly, but a case has been reported by Henry[6] in which pressure of a rudimentary left first rib on the cervical sympathetic chain and stellate

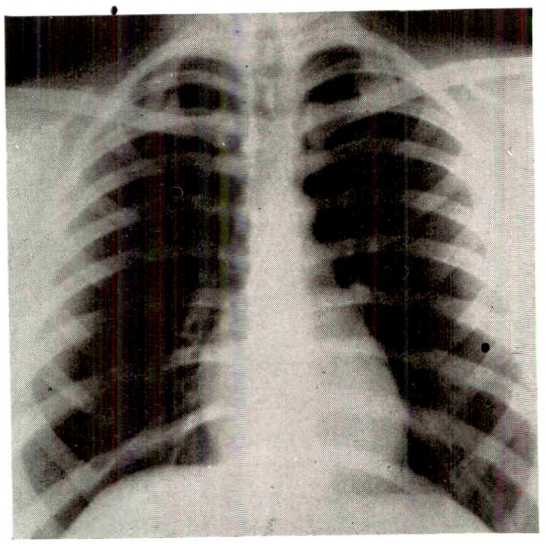


Fig. 3. Bilateral anomaly of first ribs having appearance of healed fracture or pseudarthrosis.

ganglion produced thermal changes in the left arm relieved by resection of the rib. Cases have also been reported[3,11] where aplasia of three or more ribs has caused deformities of the thoracic wall with eventration of the viscera.

Synostoses of the ribs were found more commonly on the right side than on the left side involving the right first and second ribs most frequently. It is interesting to note that synostoses were found to occur, both anteriorly and posteriorly, between all the ribs visible on the chest roentgenogram. One case only showed multiple synostoses (Fig. 4) in addition to bilateral cervical ribs, the right one of which formed

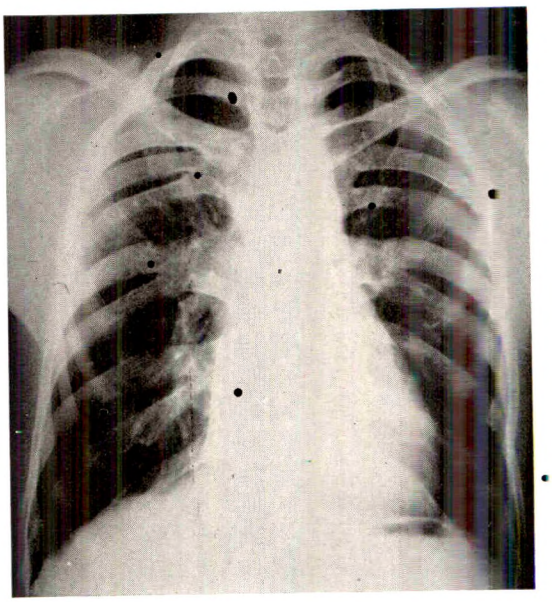


Fig. 4. Multiple synostoses and bilateral cervical ribs, the right one forming a synostosis with the first rib (pseudarthrosis).

a pseudarthrosis with the first rib. Clinically this man showed straightening of the dorsal spine with slightly limited mobility, but had no functional incapacity. An occasional synostosis had the appearance of a joint between the ribs (Fig. 5). A report of

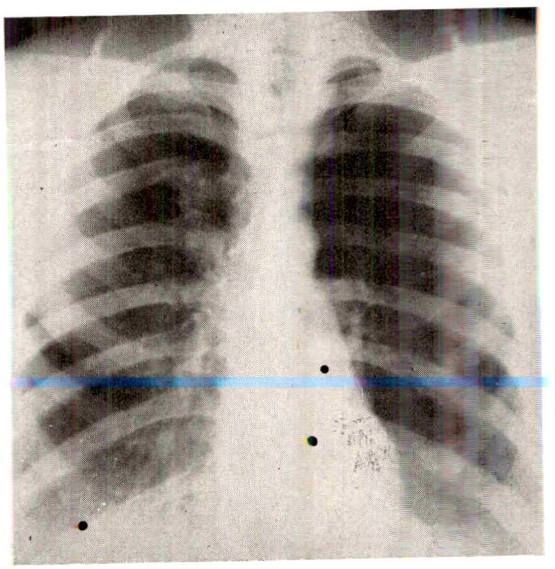


Fig. 5. Synostosis between right fifth and sixth ribs posteriorly, having appearance of a pseudarthrosis.

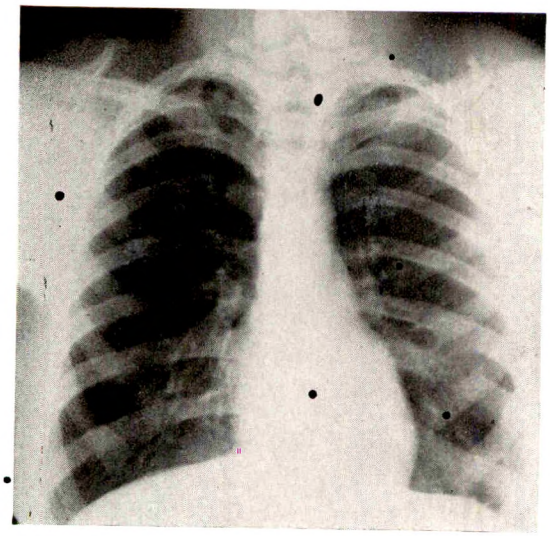


Fig. 6. Synostosis between left seventh and eighth ribs posteriorly suggesting appearance of an air cyst.

a case[8] in which there was complaint of pain from a lump beneath the left clavicle was found to be a pseudarthrosis between the end of the left first rib and the mid-portion of the second rib. Occasionally a synostosis may give rise to an appearance suggesting a pathologic process as shown in the accompanying roentgenogram (Fig. 6). Here a combination of shadows suggests an emphysematous bulla or air cyst at the left base. None of the men we saw had any symptoms or external evidence of synostosis of the ribs.

Bifid ribs showed the appearance of a lobster claw, occasionally incomplete; simple forking, and spatula forms. Their oc-

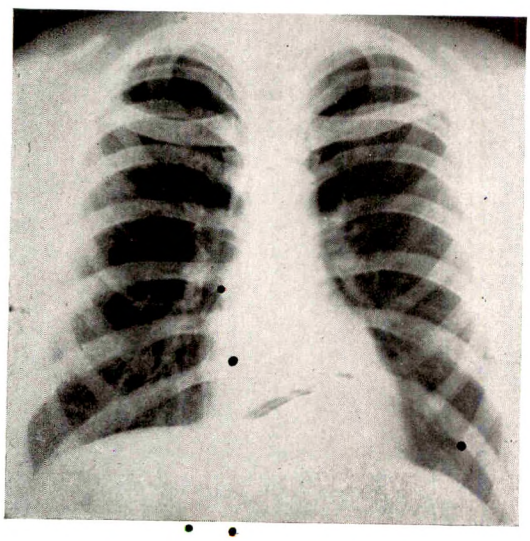


Fig. 7. Bilateral bifid third ribs.

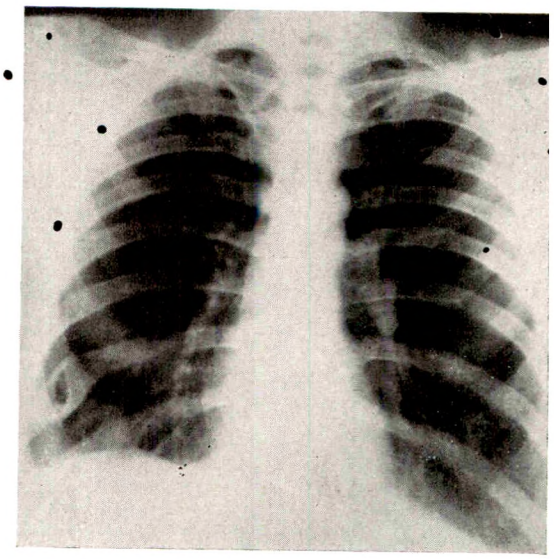


Fig. 8. Frequent appearance of rib produced by healed resection for empyema.

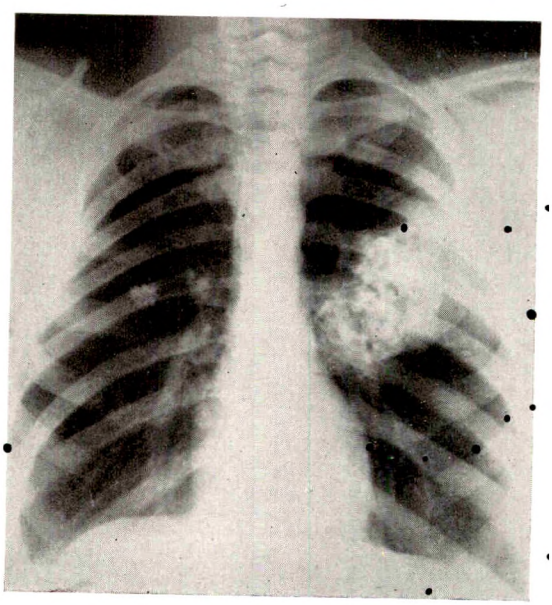


Fig. 9. Osteochondromatosis of ribs producing an appearance suggestive of massive healed primary tuberculosis.

currence was 50 per cent more on the right side than on the left side. The right fourth rib showed this anomaly most frequently. Bilateral bifid rib occurred rarely, only 7 cases being found in the series (Fig. 7).

Scoliosis of acceptable degree by Army standards; that is, less than 2 inches' deviation from the midline, on a postural basis, was noted infrequently, namely 1 in 300 men.

Healed fractures of ribs were seen only once in 800 men and ribs resected for empyema once in 1,100. The appearance produced by healed resection for empyema was fairly constant (Fig. 8) and usually showed a lacunar defect in a spindle-shaped deformity of the rib.

In one case a very unusual appearance was presented by osteochondromatosis. Upon first viewing the roentgenogram (Fig. 9) the appearance was that of a massive healed primary tuberculosis, but lateral views showed the connection anteriorly with the ribs and costal cartilages. A bone survey then was done and showed typical osteochondromata of the long bones.

SUMMARY

The statistical data of the occurrence of osseous abnormalities of the thorax have been tabulated and attention drawn to the rare conditions which may possibly be mistaken for pathologic processes.

The author expresses his thanks to Cpl. John J. White for his valuable assistance in the preparation of the statistical data.

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RATIONALE AND RESULTS OF ROENTGEN TREATMENT OF THE ADRENAL GLANDS IN ANGINA PECTORIS*†

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RATIONALE

THE syndrome of angina pectoris is generally recognized as being caused by a state of acute anoxia of the heart muscle (Keefer and Resnik, Levy, *et al.*). The presence of coronary sclerosis *per se* does not suffice to explain the characteristic acuteness of the anginal attacks.

The common conceptions of acute "coronary spasms" or acute "hemodynamic strain" are not satisfactory. Coronary spasms have never been directly observed. They were only hypothetically postulated for the purpose of explaining the clinical manifestations of acute myocardial anoxia. Moreover, such spasms are the less likely to occur, the more rigid the sclerotic coronary arteries grow. The pain-relieving effect of the vasodilating nitrites cannot be accepted as positive evidence of a preexisting spasm of the coronary vessels if the possibility of a primarily biochemical mechanism of acute myocardial anoxia is taken into consideration. Furthermore, there is no evidence of an increased hemodynamic strain as being a regular feature in angina pectoris. On the contrary, blood pressure and pulse rate may even be lower during attacks than during the interval (Raab[a]).

Shambaugh and Cutler were the first to discuss the possibility that adrenalin discharges may be involved in the pathogenic mechanism of angina pectoris. The theory advanced by one of us (Raab[b]) that anginal attacks are brought about by the acutely anoxiating effects of adrenalin discharges upon a heart muscle whose coronary arteries are sclerotic and thus unfit for adequate compensatory dilatation is based upon the following facts:

(1) Adrenalin exerts a specific, intensely anoxiating effect upon the heart muscle (Barcroft and Dixon, Evans and Ogawa, Gremels, Rein, and Green and his coworkers) which by far exceeds the oxygen requirement for the simultaneous increase of hemodynamic action (Gollwitzer-Meier, Kramer and Krüger).

(2) Adrenalin is physiologically discharged from the adrenal glands into the blood stream under the influence of physical exercise, emotions, cold (Cannon, *et al.*), under the very conditions which usually elicit anginal pain.

(3) The heart muscle possesses a greater tendency than other tissues to accumulate circulating excess amounts of adrenalin, and likewise accumulates other adrenal catechols (Raab[c,e]).

(4) During physical exercise or exposure to cold the adrenalin content of the heart muscle increases (Fig. 1) while that of the adrenal glands diminishes (Raab[c,e]).

(5) Injection of adrenalin is likely to produce anginal symptoms in persons with coronary sclerosis (Levine, Ernstene and Jacobson; Katz, Hamburger and Lev; Cottrell and Wood) and, if given in large doses, also in healthy persons (Wenckebach, (Raab[b], self-experiment).

(6) Abnormally intense discharges of adrenalin into the blood stream during physical exercise were observed in all of 12 patients suffering from angina pectoris. They disappeared in all of 7 successfully treated patients in whom the tests were repeated after treatment (Raab[d]).

(7) The electrocardiographic changes which occur during anginal attacks are practically identical with those following the administration of adrenalin (Vesa,

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Lepeschkin, Katz, Hamburger and Lev, *et al.*).

(8) Patients with tumors of the adrenal medulla often suffer from anginal attacks (Bauer, Scherf, Nuzum and Dalton, Brunschwig, Humphreys and Roome, *et al.*) during which an increased adrenalin level was found in the blood (Brandt and Katz) and which disappear after removal of the adrenal tumor (Lenhartz).

(9) Conditions which are known to increase the secretion of adrenalin, such as tobacco smoking (Cannon, Aub and Binger; Eichholtz; Stewart and Rogoff; Kobro; Raab[1]) of overdosage of insulin (Brandt and Katz; La Barre and Houssa; Kugelmann; Raab[1]) or breathing of low oxygen concentrations (Cannon and Hoskins) are likely to elicit anginal symptoms (Külbs; Deneke; Strauss; Hadorn; Schönbrunner; Rothschild and Kissin; Levy, *et al.*).

Large doses of roentgen rays applied to the adrenal region of animals have been found to be followed by structural changes of the adrenal glands (Harvey; Cottenot; von Decastello; Grabfield and Squier; Edlfelder and Peiper; Martin, Rogers and Fisher; Tsuzuki; Desjardins; Engelstad and Torgersen). These changes concern chiefly the cortex, although the medulla was also found to participate in part of the experiments.

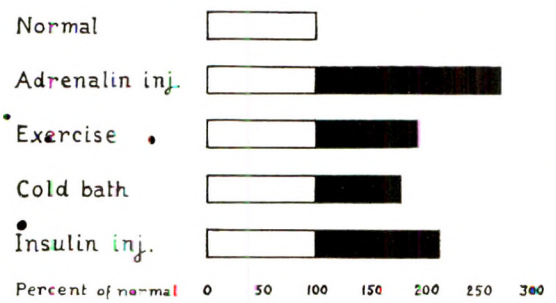


Fig. 1. Concentration of adrenalin and adrenalin-like substances in the heart muscle of the white rat. The white squares indicate the average normal concentration per gram of myocardial tissue; the black areas indicate the increase of hormonal material beyond normal under experimental conditions: Subcutaneous injection of 2.5γ of adrenalin per gram body weight; swimming in a pool for six to fifteen minutes; a cold bath of 5° C. for ten minutes; subcutaneous injection of an average of eight units of insulin.

Recent experiments carried out by us on rats showed that single doses of 100 and 1,000 roentgens caused only slight changes

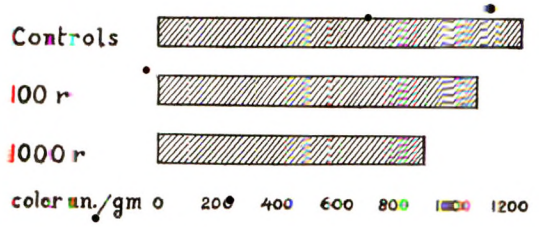


Fig. 2. Adrenalin plus adrenalin-like substances in the heart muscle of the rat following physical exercise (swimming in cold water). Two months after irradiation of the adrenal region with 100 and 1,000 r respectively the total concentration of the hormonal substances in the heart appears diminished, probably due to diminution of the excess influx of this material into the heart muscle which is normally taking place during exercise (compare Fig. 1) (Raab and Soule).

of the content of the adrenal glands in adrenalin and related catechols within two months. However, the accumulation of such hormonal material in the heart muscle following physical exercise was distinctly diminished two months after irradiation of the adrenal region (Fig. 2).

Desjardins, Frey, and Engelstad and Torgersen, in contrast to earlier investigators, reached the conclusion that the roentgen-ray sensitivity of normal adrenal glands is not greater than that of other tissues. However, the roentgen-ray sensitivity of living cells increases with the degree of their activity (Holzknecht's law) and the state of overactivity of the adrenal medulla and of its nervous secretory apparatus which seems to prevail in angina patients, as discussed above, makes the possibility of a beneficial, normalizing effect of roentgen treatment upon the adrenal glands of angina patients and upon their nervous supply plausible, even with a dosage which does not suffice to cause any damage to the glands.

The results of irradiation of the adrenal glands in 100 European patients with an-

TABLE I

IMPROVED CASES

Case No.	Age	Sex	Blood Pressure	Severity	Duration (yr.)	Electrocardiogram	Coronary Thrombosis (years ago)	Complications	Date of Treatment	Improvement		Other Medication after Roentgen Treatment
										Degree	Duration (mo.)	
1	54	♂	170/100	M	½	−	—	—	7/39	+++	45	Quinidine
2	47	♀	100/70	M	2½	+	—	—	10/39 5/40	++	32 11	—
3	50	♀	216/98	M	2	+	1	Spinal arthritis +	9/39	+++	42	—
4	58	♂	142/80	S	1/6	+	—	Temporary decompensation	10/39	+++ − +++	30 (1) 19	Digitalis
5	63	♂	160/80	M	8	−	—	—	5/40	++	36	—
6	54	♀	200/100	M	1	?	—	Temporary decompensation. Died 11/43	4/40 12/40	++ − + −	7 (2) 20 (13)	Digitalis
7	69	♂	118/80	M	3	+	—	Temporary decompensation. Br. asthma	4/40 1/41	++ ± +++	5 3 32	Digitalis
8	60	♂	175/96	M	5	++	—	Spinal arthritis ++	12/40 7/42	+++ ± ++ +++	12 1 6 17	—
9	47	♂	155/100	S	¾	+	—	Spinal arthritis +	12/40 4/41 +Th.*	+ +++	4 26	—
10	35	♂	166/78	L	1½	++	—	Hyperthyroidism	2/41	+++ ++ +++	11 7 8	—
11	71	♂	126/55	S	6	++	2½	—	2/41* 4/41* 11/41	+ ++ ++	2 7 18	Theocalcane
12	53	♀	190/106	M	14	+	14; 4		3/41	+++	26	—
13	59	♂	150/70	S	3	+	3	Temporary decompensation. Died 8/42	10/39 7/41	++ − ++ −	9 (11) 5 (9)	Digitalis
14	57	♀	170/86	S	2	++	—	Pernicious anemia. Spinal arthritis ++	6/41 8/41 +Th.	+ ++ +++	2 9 12	Liver extract
15	67	♂	210/110	S	6	?	—	Temporary decompensation Spinal arthritis +	9/39 4/40	++ − +++ ±	6 (1½) 6 4	Digitalis
16	72	♂	156/80	M	6	+	—	—	3/41 12/41*	++ +	8 18	—
17	55	♂	148/86	M	1½	+	—	Spinal arthritis +	12/40* 2/41* 9/41 +Th.	− + +	(4) 13 10	—
18	56	♂	170/110	S	10	+	—	—	12/40 3/41	++ +++ ++	2 18 9	—

TABLE I—(*Continued*)

Case No.	Age	Sex	Blood Pressure	Severity	Duration (yr.)	Electrocardiogram	Coronary Thrombosis (years ago)	Complications	Date of Treatment	Improvement: Degree	Improvement: Duration (mo.)	Other Medication after Roentgen Treatment
19	60	♂	160/88	L	4	+	—	—	3/41 3/42*	+++ ± +++	8 3 9	—
20	60	♂	160/86	S	½	—	½	Meteorism	7/41	+++ ++	2 20	—
21	56	♂	180/110	S	2½	+	½	Aerophagia Died 2/43	5/41 8/41*	– + ++ +++	(3) 4 6 8	Theobromine, quinidine
22	51	♀	166/80	S	2	+	—	Diabetes	7/41 9/41*	++ ++	2 20	—
23	72	♂	160/80	M	⅔	++	—	—	7/41	++ +	2 20	—
24	61	♂	160/90	M	12	?	—	—	5/40	+++ –	16 25	—
25	60	♂	140/100	M	3	+	—	—	7/41	++	22	Thiamine
26	58	♂	140/100	S	4	+	1½	Spinal arthritis +	10/41 7/42	++ ++	7 11	—
27	75	♀	176/90	M	3	+	2	Spinal arthritis +	12/41	+++ ± +++	6 2 16	Aminophylline
28	62	♂	130/82	M	1½	++	—	Died 8/41	1/41	+++	7	—
29	64	♀	220/112	S	2	++	—	Died 2/42	2/41	++ ?	5 (7)	—
30	63	♂	186/100	M	5	++	5	Died 2/43	3/42	++ +	4 2	—
31	75	♂	198/118	M	3	+	—	—	10/41 6/42	— ++	(7) 18	Theocalcine

S = severe; M = medium; L = light.
* Treatments carried out in other places with supposedly the same technique.

gina pectoris have been published previously by one of us (Raab[b]). Seventy-six of these patients were either entirely freed of their complaints or at least improved for an average period of twelve and a half months, calculated until the time when further follow-ups became impossible for technical reasons.

In this country 42 typical angina cases have been examined and treated by us since July, 1939. Thirty-one of these were males, 11 were females.

As shown in Tables I and II, the cases were classified as severe (S: daily intense attacks brought about by slight exertion, etc., and also occurring at rest), medium (M: attacks less frequent or less intense and uncommon at rest) and light (L: attacks occurring only now and then or of a mild degree).

The technique of treatment consisted in a series of six single treatments on consecutive days; three to the left upper quadrant of the abdomen, posteriorly, and three to the right, the left and right areas being treated on alternate days. Two hundred roentgens, measured at the skin, were administered at each treatment. Thus, each

TABLE II

UNIMPROVED CASES

Case No.	Age	Sex	Blood Pressure	Severity	Duration (yr.)	Electrocardiogram	Coronary Thrombosis (years ago)	Complications	Date of Treatment	Improvement: Degree	Improvement: Duration (mo.)	Other Medication after Roentgen Treatment
32	48	♂	140/100	S	8	++	—	—	3/41 6/41* 8/41*	— + —	(5) 2 (2)	—
33	66	♀	170/90	S	5	++	5 ½	Spinal arthritis ++	3/41 5/41 10/41 +Th.	— + —	(5) 1 (7)	—
34	60	♂	132/80	M	10	+	10 ½	—	11/41 3/42*	— —		—
35	34	♀	250/170	S	2½	+	—	Temporary decompensation	3/41 5/41	— —		Digitalis
36	58	♂	212/84	S	6	—	—	—	4/40 8/40	— —		—
37	63	♂	170/110	M	½	+	—	Spinal arthritis ++ Died 2/42	9/41 12/41	+++ + —	1 1 (2)	Theobromine
38	66	♂	126/66	M	4	—	—	—	3/41	+ —	2 (12)	—
39	52	♂	118/74	M	4	—	—	—	1/41	—		—
40	47	♀	124/62	M	4	+	—	—	3/41	++ —	1 (3)	—
41	64	♂	230/130	L	10	+	—	Paroxysmal tachycardia	1/41	—		—
42	55	♂	130/76	S	8	++	—	Thyroidectomy 6 yr. ago (B.M.R. −19%)	10/40	—		Thyroid

* Treatments carried out in other places with supposedly the same technique.

area was given 600 r as a total dose per series. The other technical factors were: 200 kv. (peak), 20 ma., 50 cm. target skin distance, 1.5 mm. Cu plus 1.0 mm. Al filter; size of fields 15 by 15 cm.

Several patients with evidence of associated hypertrophic spinal arthritis were given supplemental irradiations to the thoracic and cervical spine, a single dose of 200 r being administered to each involved area through a 10 by 15 cm. cone.

In a number of instances the series of treatments over the adrenal region was repeated either by the one of us (Soule) or by other roentgenologists in other places. When the latter was the case, it is indicated by asterisks in Tables I and II. The minimum intervals between two successive series of treatments were six to eight weeks.

RESULTS

Complete disappearance or marked improvement (+++) of the anginal symptoms for six to forty-five months occurred in 38 per cent of the 42 cases; another 33 per cent were considerably improved (++) from five to forty-three months; 3 per cent were only slightly improved (+), and 26 per cent remained unimproved (−). Among the latter 11 cases only 2 had received the full three series of treatments, and thus are to be qualified as definite failures.

In the improved cases the average number of series of treatments given was 1.6.

The duration of uninterrupted improvement ranged between five and forty-five months, with an average total duration of twenty-four months, up to date. Temporary complete relapses with the symptoms as intense as before treatment occurred in sixteen per cent of the improved cases, and complete relapses still continuing at the time of the patient's last report or ending in death in 10 per cent.

Blood pressure, electrocardiogram and other clinical features could not be followed as carefully as in the European material since most of the patients discussed in the present series had come for treatment from distant places and remained in contact with us only by correspondence.

In 8 hypertensive cases there was no significant change of the blood pressure despite a marked improvement of the anginal symptoms (average 175/81 before and 184/83 mm. Hg after treatment). The same had been true in the hypertensive European patients whose anginal complaints were improved.

In 6 cases a pathological electrocardiogram at rest showed partial or complete normalization. Electrocardiograms after exercise were not carried out in this series.

Eleven of the 31 improved cases continued the use of various drugs after the roentgen treatment, as indicated in Table 1, for varying periods of time.

In 7 cases in whom abnormally intense discharges of adrenalin proper and adrenalin-like substances (catechols) into the blood stream had been observed in connection with physical exercise, these discharges disappeared as soon as clinical improvement had set in (Fig. 3) and reappeared in 1 case during a relapse.

Death occurred in 7 of 42 patients treated within a period of four and a half years. Three of these (No. 21, 28 and 30) died suddenly during a state of clinical improvement, apparently from coronary occlusion; 2 cases (No. 21 and 28) had been practically free of anginal complaints for periods of several months until death. Three others (No. 6, 13 and 29) died during relapses of their anginal condition after temporary improvement; 1 patient (No. 37) who had been classified as unimproved died from pneumonia. In Case 13 an autopsy was performed. The medium sized coronary branches were sclerotic. The adrenal glands appeared normal grossly. Microscopically "the adrenal cells were well preserved. The cells of the zona fasciculata and the zona reticularis appeared unusually foamy." The kidneys showed signs of passive congestion and moderate atheromatosis of the arterioles.

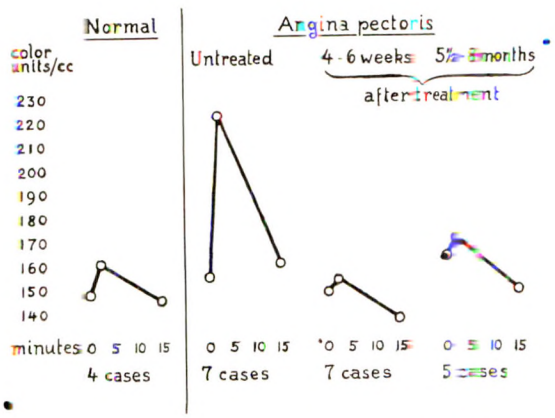


FIG. 3. Reaction of the blood level of adrenalin and adrenalin-like substances to a standard physical exercise test (climbing stairs) in normal persons and in angina patients before and after roentgen irradiation of the adrenal region (average curves).

(In a previous publication (Raab[4]) the figures given in these curves were interpreted as representing "adrenocortical compounds." Later investigations have shown, however, that cortical steroids do not participate and that the chromogenic material which is expressed in color units per cubic centimeter of blood consists essentially of adrenalin proper and of adrenalin-like catechols (Raab[6]).)

One patient who is not included in Table 1, a man aged forty, with severe attacks of only a few months' duration following a coronary occlusion, died two weeks after the first series of treatments from an occluding hemorrhage under the intima of the right coronary artery. The adrenal glands appeared normal at autopsy.

Most of the patients from out of town were questioned about their condition at repeated intervals. Since the subjective

Table III

	Patients Treated in Burlington (1939–1942) 42 cases		Patients Treated in Vienna (1936–1938) 100 cases	
	Improved	Unimproved	Improved	Unimproved
Number	74%	26%	76%	24%
Age	59 yr.	55 yr.	60 yr.	60 yr.
Males Females	74% 26%	73% 27%	84% 16%	87% 13%
Pathological electrocardiogram at rest	77%	73%	50%	30%
Coronary infarction before treatment	22%	18%	6%	1%
Spinal arthritis	22%	18%	20%	21%
Severe cases Medium cases Light cases	39% 55% 6%	46% 46% 8%	28% 58% 14%	50% 38% 12%
Duration of angina pectoris before treatment (average)	4 yr.	5½ yr.	3¼ yr.	5⅓ yr.
Systolic blood pressure (average)	164 mm.	164 mm.	154 mm.	172 mm.
Average duration of improvement	24 mo.	—	12½ mo.	—
Reported cardiac deaths*	14%	0%	1.3%	4%

* The discrepancy between the percentages of reported cardiac deaths in the American and the European cases may be due in part to the shorter total period of contact with the latter and the lack of further reports about their fate.

reaction of angina patients is the most decisive criterion of therapeutic results, some of the written answers received will be quoted in brief abstracts (the first and, if any, the second and third series of treatments will be indicated as *I, II, III*):

Case 1 (M): 34 months after *I:* "I can walk on even level. Cold weather does not affect me. . . . There is no comparison how much better I feel now than before treatment."—38 months after *I:* "I am happy to state that my last report still holds good."—46 months after *I:* "No change."

Case 2 (M): 32 months after *I:* "I feel very much better than before treatment but my breathing is very short. . . . I have made my own garden this year."—43 months after *I:* "I walk good now but slow, am better than before treatment."

Case 3 (M): 33 months after *I:* "Feeling fine and am not bothered with any pain."—43 months after *I:* "Feeling fine, everything OK."

Case 5 (M): 25 months after *I:* "If I move slowly and take my time in my work, my chest gives little trouble . . . cold weather didn't seem to affect the pain. . . . The treatment did give considerable relief."—37 months after *I:* "Am at present working every day, do not feel any pain unless I hurry. In cold weather I still get pains. . . . Condition better than before treatment."

Case 7 (M): 26 months after *I:* 16 after *II:* "I have no chest pain. . . . A great deal better than before treatment."—42 months after *I:* 32 after *II:* "Continually free of chest pain."

Case 8 (M): 19 months after *I:* "I rarely ever have any chest pains or distress, except when I indulge in over-eating. . . . It is my impression that the treat-

ments have given me great relief."—36 months after *I;* 17 months after *II:* "Glad to say that I am well."

Case 9 (S): 17 months after *I;* 13 after *II:* "No chest pain or distress, can walk and do other routine physical exercise." Work involved "continual walking, chopping brush, etc., and it was all carried out without distress."—30 months after *I;* 26 after *II:* "Improvement almost 100 per cent."

Case 10 (L): 16 months after *I:* "Decidedly improved. . . . Am able to do the routine work as a janitor but not to shovel snow, carry heavy loads up stairs, etc."—26 months after *I:* "No distress excepting one day when engaged in unusual physical activity. . . . Greatly improved."

Case 11 (S): 16 months after *I;* 7 after *III:* "I have been greatly improved. I only take a nitroglycerine tablet once or twice a month. I am able to do routine household duties with much greater ease. I can't walk well in windy or cold weather."—27 months after *I;* 18 after *III:* "I feel that my condition is good. . . . Much improved but still chest pain from cold and windy weather. Over-eating causes distress."

Case 12 (M): 15 months after *I:* "There is no comparison with the condition before treatment. . . . I find it a pleasure to walk and am able to climb stairs and come up the hill without any after effects. I am now able to do almost everything."—26 months after *I:* "Even further improved. Have been able to do all things with more ease."

Case 14 (S): 13 months after *I;* 11 after *II:* "Very much improved. . . . No chest pains. Am able to walk short distances, also to do my few household tasks. . . . A great deal of relief up to and including the present time."—23 months after *I;* 21 after *II:* "Much the same; much better than before treatments."

Case 16 (M): 13 months after *I;* 4 after *II:* "The treatments have given considerable relief. . . . I walk a mile or more each day. . . . Reactions to emotions and cold not so good. . . . Am decidedly better than before treatment."—26 months after *I;* 17 after *II:* "Not much further change." Altogether "considerably improved."

Case 17 (M): 16 months after *I;* 14 after *II:* "My condition is improved. . . . Still have daily manifestations, even though mild, of chest distress brought on by walking."—27 months after *I;* 25 after *II;* 18 after *III:* "No further change."

Case 18 (S): 12 months after *I;* 9 after *II:* "I feel a whole lot better than it was before the two treatments. I hunted deer five days and ran a trap line for three weeks . . . and have done a lot of walking and mountain climbing. . . . I couldn't have done these things a year ago or a tenth part of it."—29 months after *I;* 26 after *II:* "Working 60 hours a week on defense work in a machine shop. . . . Can walk miles if do not eat. . . . Most distress after supper. Walking uphill bad but very much better than before treatment."

Case 19 (L): 15 months after *I;* 3 after *II:* "I am infinitely better than before the treatments. They have greatly relieved my chest complaint. I can walk a dozen squares now . . . when I could not walk two before."—21 months after *I;* 9 after *II:* "Still much benefited by the treatment. . . . Have reactions after effort but as long as I am careful I am wonderfully well."

Case 20 (S): 11 months after *I:* "Condition better than before treatment. . . . At times there is a recurrence of the pains in the left shoulder but not nearly so frequently nor so severe."—22 months after *I:* "The same or perhaps slightly better." Altogether "most decidedly improved."

Case 21 (S): 13 months after *I;* 9 after *II:* "Am much better. . . . Attacks of chest pain increasingly fewer and much milder. I have had as much as two weeks on end of freedom from pain. Discomfort and pain spells after meals have almost completely disappeared. . . . Feel very happy about the wonderful improvement."—Died suddenly after 8 more months without attacks (according to letter from his widow).

Case 22 (S): 11 months after *I;* 9 after *II:* "My condition is much better than before treatment. . . . I just have a little trouble getting undressed and to lie down in bed. Just for a minute."—22 months after *I;* 20 after *II:* "I am doing swell. I can walk up and down stairs without trouble. . . . The treatments have given me wonderful relief."

Case 23 (M): 11 months after *I:* "Am much better than before the treatment. Can do light work if I start off gradually. Can walk leisurely, but not far at a time, without stopping to rest when I feel the chest pains."—22 months after *I:* "Much better than before treatment but cannot walk much or do anything strenuous, especially if cold and windy."

Case 25 (M): 10 months after *I:* "I am doing much better this year than last. I can walk better, eat better and do my work much easier."—22 months after *I:* "Better than before treatment, but cold bothers and pain if I exercise after meals."

Case 26 (S): 7 months after *I:* "I am much better. . . . Pain improved considerably. I can walk about a block before I begin to get pains. . . . Emotions, cold weather and meals seem to be about the same as before."—18 months after *I;* 10 after *II:* "Am suffering much less pain in chest."

Case 28 (M): 8 months after *I:* "I am working five days a week and am feeling very well. I walk a couple of miles a day and feel fine for it but a couple of times when the wind was blowing hard I felt a slight pain but it left right away." (Climbs 22 steps every hour 40 times a week).—Died suddenly one month later.

Case 29 (S): 5 months after *I:* "I am feeling much better now and by being careful do not suffer much pain."—Died 7 months later.

Case 30 (M): 4 months after *I:* "I am distinctly improved regarding pain. My ability to walk is distinctly improved."—7 months after *I:* "Still definitely better than before radiation therapy. . . . However pain of moderate severity is becoming more easily produced either by exertion or from emotional stress."—Died 5 months later.

Case 31 (M): 3 months after *II:* "Decided relief from chest complaints. . . . I have less pressure and with a pause now and then things get normal."—18 months after *II:* "Feel fairly good. Got employment as messenger, am out of doors quite a bit."

Nine patients who had been incapacitated through their anginal symptoms resumed work: 2 as farmers (No. 5 and 7), 1 as a foreman in a factory (No. 28), 1 as a restaurant chef, later going into defense work (No. 18), 1 as a cook (No. 6), 1 as a civil engineer (No. 9), 1 as a minister (No. 21), 1 as a bookkeeper (No. 17) and 1 as a messenger at the age of seventy-six (No. 31).

DISCUSSION

The results of roentgen irradiation of the adrenal glands in angina pectoris in the above described series of American patients are similar to those obtained in Europe by one of us (Raab[b]) and by other investigators.

Hadorn (Berne) applied the method "in 12 cases of ambulatory angina pectoris, partly of the severest degree, which had been treated unsuccessfully for a long time. The effect was an immediate one in 7 cases." In 1 case a normalization of the electrocardiogram which had been persistently pathological for four years, accompanied the subjective improvement.

Lippross (Dresden) reports 4 cases with the conclusion: "In severest cases of status anginosus we achieved the best results with roentgen-ray irradiation of the adrenal glands as suggested by Raab."

Professor A. Schittenhelm, head of the Second Medical Clinic of the University of Munich writes: "My experiences are only limited but still of a kind that I would recommend under all circumstances radiation therapy to every patient with angina pectoris. . . . It would have to be applied over both adrenal glands and possibly also over the cervical and upper thoracic portion of the sympathetic system." According to a personal communication received in 1941 from Professor Schittenhelm, several of his successfully irradiated patients had remained free from relapses for periods of two years.

In this country the disappearance of precordial distress in 5 patients treated for arterial hypertension with combined irradiation of the pituitary and the adrenal glands was mentioned as early as 1935 by Hutton.

Graybiel in a review on "Diseases of the Heart" makes the following comment on the observations of one of us on 100 cases (Raab[b]): "We are frankly skeptical of these results, having used a similar procedure in a small number of cases without success." In a personal letter Dr. Graybiel mentions an improvement in "two or three" patients which "might well have come within the realm of individual variability." Apparently only 400 r and only one single series of treatments had been applied in his patients.

Untoward side-effects were never observed by us or mentioned by any one of the above named investigators, except a lack of appetite or moderate nausea in about 15 per cent of the cases, lasting for only a few days. No signs suggestive of adrenal insufficiency were ever observed. Thus, the warning with which a number of patients were cautioned against the treatment that it is "dangerous" and "likely to produce Addison's disease" appears entirely unfounded.

In none of 7 patients in whom the adrenal hormone level in the blood at rest was determined did it fall below normal limits after roentgen treatment of the adrenal glands (Raab[d]). In 3 irradiated cases (one of them in Europe) who had died, no significant structural changes of the adrenal glands were found at autopsy, except "un-

usually foamy cells" in the cortex of Case 13, more than one year after treatment when he died of cardiac failure. The adrenal cells were "well preserved" in this case.

The essential effect of the treatment seems to consist in the abolition of abnormally intense adrenalin discharges on effort, etc., through suppression of an existing state of abnormal irritability of the nervous mechanism of adrenal medullary secretion. The abnormally intense discharges on physical exercise disappeared in all of 7 patients who were subjected to this test after successful therapeutic irradiation of the adrenal glands (Fig. 3). These observations tend to support the conception of Celotti and Gouin and Bienvenue that moderate roentgen-ray doses applied to the adrenal region exert their functional effects mainly by influencing the supplying nervous sympathetic structures and through "restoration of functional equilibrium."

While the rationale of the treatment in question makes its beneficial effects easily intelligible, it appears more difficult to explain the failure to respond of a substantial percentage of cases. Insufficient dosage alone can hardly be made responsible in a fraction of these: 12 of the 24 unimproved patients of the European series and 2 of our 11 unimproved American patients had received the full dose of three series, or even more, without effect.

Attempts to detect any clinical characteristics of the non-responding patients regarding age, sex, electrocardiogram, past myocardial infarctions and other complicating factors, such as hypertrophic spinal arthritis, were to no avail (Table II). Only a somewhat longer average duration and greater severity of the disease before application of treatment in the non-responding patients seemed to be of a certain, though limited, significance.

The fact that adrenalin is formed not only in the adrenal glands but also by sympathetic nerve fibers in the peripheral vascular system and in the heart muscle itself (Cannon and Lissak) and that stimulation of these nerves increases the myocardial oxygen consumption in the same manner as adrenalin does (Gollwitzer-Meier, Kramer and Krüger) suggests the possibility that a prevalence of this local "adrenergic" mechanism within the heart muscle makes roentgen irradiation of the adrenal glands alone insufficient in certain cases.

The observations of Desjardins and others in regard to a specific depressant effect of roentgen rays upon the irritability of nerve cells may furnish the explanation for the beneficial action of paravertebral and precordial irradiation in angina pectoris as described by Beeck and Eirsch, Morlet, Torbett, Lian, Nemours-Auguste and Barrieu, Gilbert, Arrillaga, Sussman, Samuel and Bowie, Holmes, and Vasch and Schenck.

In our own material paravertebral cervical and thoracic irradiations were added to the adrenal treatments in 4 cases with hypertrophic spinal arthritis. In 2 of these cases this combined form of treatment seemed to improve the results (Cases 9 and 14). The same had been true in 6 of 18 patients treated in Vienna in a similar manner.

The fundamental importance of hormonal cardiometabolic factors in the pathogenesis of angina pectoris is evidenced by a variety of facts: males are very much more afflicted with this disease than females. It becomes apparent, as a rule, at the age of sexual involution. In castrated animals the adrenalin concentration of the heart muscle is increased (Raabe), and the latter displays metabolic alterations which are identical with those elicited by adrenalin (Schumann). These changes are normalized through the administration of sex hormones (Schumann). Favorable therapeutic results with sex hormones in angina pectoris were reported by Henssge, Pick, Arndt, Scherf (including normalization of the electrocardiogram), more recently also in this country by Walker, Bonnell, Pritchett and Rardin, Lesser, Hamm, McGavack. Also thyroidectomy has proved to be of therapeutic value in angina pectoris (Lev and Hamburger, Blumgart, Levine and

Berlin, Claiborne and Hurxthal, Clark, Means and Sprague, *et al.*). Since adrenalin was found to accumulate in the heart muscle in maximum concentrations and with maximum speed under the influence of the thyroid hormone (Raab[c]) it seems that the therapeutic effect of thyroidectomy consists essentially in a protection of the heart against the influx of high adrenalin concentrations.

Against the assumed value of roentgen treatment of the adrenal glands for angina pectoris the objection has been raised (von Zimmermann-Meinzingen) that the improvements observed might be of a merely psychic nature. Although such factors may play a rôle in some of the improved patients, it seems difficult to attribute to them a general importance for the following reasons:

(1) It is unlikely that purely imaginary improvements of previously stubbornly intractable anginal attacks should persist uninterrupted for many months and even over periods of years. Only 4 out of 23 improved European patients who were exposed to extreme excitement and fear suffered permanent relapses.

(2) In many cases the improvement was not felt earlier than several weeks after the treatment or after a second or third series. One would expect a purely psychic reaction to take place immediately when the patient is still under the fresh impression of the procedure.

(3) The chemically demonstrable disappearance of the causative abnormal adrenalin discharges during exercise (Fig. 3) and the normalization of exercise electrocardiograms (Raab and Schönbrunner) after the treatments are objective criteria of improvement.

More important seems the possibility of spontaneous improvements and recoveries which are not uncommon in angina cases as pointed out by Levine and White. However, it appears extremely improbable that such spontaneous turns toward the better should have occurred and persisted in 107 out of 142 patients and that they should have coincided with the time of treatments as closely as can be seen from the graphic "complaint curves" (Raab[b]).

Nevertheless it would be highly desirable to have extensive series of both real and sham treatments carried out in medical centers with a large out-patient material and to have the results followed and analyzed in a critical and unbiased manner, not only for practical reasons but also for the purpose of throwing more light upon the problem of the biochemical origin of the angina pectoris syndrome.

It should be re-emphasized in this connection that seemingly non-responding patients who have received less than three series of irradiations within about one year must not be classified as definite failures since one or even two series prove not infrequently an insufficient dose.

The occurrence of a coronary occlusion is to be regarded as a definite contraindication to the treatment for a period of two to three months. An existing state of cardiac decompensation should be controlled with digitalis, and so forth, before treatment is begun. Patients with arterial hypotension or with tuberculosis should not be given roentgen treatment.

SUMMARY

Rationale. Angina pectoris on effort, emotion, and so forth, is believed to be due essentially to the well known acutely anoxiating effect of adrenalin discharges from the adrenal glands upon the heart muscle, the sclerotic coronary arteries of which are unable to dilate adequately and thus to overcome the resulting myocardial anoxia. Clinical and experimental evidence is presented for this theory.

The state of abnormal irritability of the adrenal secretory mechanism which was found to be a characteristic of angina patients could be abolished through roentgen irradiation of the adrenal region without ensuing damage to the basic normal function of the glands. This was demonstrated objectively by quantitative chemical hormone determinations in the blood before

and after treatment and by the normalization of pathological electrocardiograms.

Therapeutic Results. Of 42 typical angina patients treated in Burlington, Vermont, with irradiation of the adrenal region, 74 per cent were either completely freed of their anginal complaints or at least improved for periods ranging between five and forty-five months with an average of two years up to the present.

Nine of the 11 unimproved patients had not received three series of roentgen treatments and thus cannot be considered as definite failures.

No untoward side effects were noted except short episodes of nausea in a few cases.

Death occurred in 7 out of the 42 patients treated during the past four and one-half years.

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Twenty-eighth Annual Meeting: 1944, to be announced.

was what, with our present methods of analysis, we would consider to be within normal limits. The question arose whether or not the heart would exhibit a similar behavior.

To answer this question, Leach and Sugiura[3] in a first series of experiments accurately established the physical magnitude of the dose, expressed in roentgens, that can be administered to the heart of rats to produce a recordable immediate effect. They used roentgen rays obtained with 200 kv., 0.5 mm. Cu, and irradiated the heart through a precordial window 1 by 1.5 cm. in size, the entire animal being covered by lead. Since the fractionated protracted method of irradiation would be most difficult to carry out, large single doses of 1,500, 3,000, 5,000, 7,500, 10,000, and 20,000 r were given. The results of these experiments supported the conclusions of previous investigators that the heart is extremely radioresistant. The critical dose for the myocardium was found to be 10,000 r or more, when capillary hemorrhage, round cell infiltration, myocardial degeneration and necrosis followed. The pericardium and endocardium withstood doses up to and including 20,000 r.

In their second series of animal experiments, Leach and Sugiura[4] extended the period of observation to from two to fourteen months after irradiation to determine if any late changes, particularly fibrosis, could be detected. Domagk[2] in his experiments found that fatty degeneration and necrosis was followed, after a time, by the interstitial proliferation of fibroblasts and therefore it was important to clarify this point. The irradiation was carried out with a technique identical with that of the first series and the doses were of the same magnitude. The results revealed that there was no demonstrable effect on the heart of rats using single doses up to and including 7,500 r. As already stated, 10,000 r represents the critical dose for the myocardium. If one could get a sufficient number of animals to survive such doses, it is likely that the immediate effect would give place to fibrosis of the myocardium, thus supporting the observations of Domagk.

In extending these experiments, Leach and Sugiura further found that no late changes in the heart of rats could be demonstrated when the roentgen rays were administered over the lungs or thigh muscle and that pulmonary infection, when present, likewise remained without effect on the heart. To explain the extremely high radioresistance of the heart, as compared to other structures of the body, Leach and Sugiura accept the fluid flow theory of Failla.[5] They believe that it is compatible with this theory to assume that the myocardium can cope with the increased ionic concentration produced by the roentgen rays up to 7,500 r but above that the intracellular concentration becomes too high to be consistent with normal metabolism of cell life.

In coordinating the above experimental data with clinical facts, Leach and his associates made some interesting new observations. A systematic study of several groups of patients since 1938 revealed in numerous instances definite disturbances of the heart and circulation following roentgen therapy for the treatment of cancer of different regions of the body. At first glance, this appeared to contradict the experimental results and therefore investigations in other directions were in order. Leach, Farrow, Foote and Wawro[6] made an exhaustive study of the fibrosis of the lung occurring following roentgen irradiation for cancer of the breast. Soon afterward, Leach[7] published his novel interpretation of the pulmonary physiology in chronic irradiation

[3] Leach, J. E., and Sugiura, K. Effect of high voltage roentgen rays on the heart of adult rats. Am. J. Roentgenol. & Rad. Therapy, 1941, *45*, 414–425.

[4] Leach, J. E., and Sugiura, K. Late effect of high voltage roentgen rays on the heart of adult rats. Am. J. Roentgenol. & Rad. Therapy, 1942, *48*, 81–87.

[5] Failla, G. Theory of biological action of ionizing radiations. *Cancer Probl., Symposium*, 1937, pp. 202–214.

[6] Leach, J. E., Farrow, J. H., Foote, F. W., Jr., and Wawro, N. W. Fibrosis of the lung following roentgen irradiation for cancer of the breast. Am. J. Roentgenol. & Rad. Therapy, 1942, *47*, 740–747.

[7] Leach, J. E. Abnormal pulmonary physiology as a result of chronic irradiation pleuropulmonitis. Am. J. Roentgenol. & Rad. Therapy, 1943, *50*, 772–778.

VOL. 51, NO. 3

EDITORIAL

EFFECT OF ROENTGEN THERAPY ON THE HEART

IN 1932, Desjardins[1] made an exhaustive review of the medical literature dealing with the action of roentgen and radium rays on various organs of the body. He found all the evidence available at that time pointing to the fact that the specific radiosensitivity of the heart is definitely less in comparison with that of most of the other organs and that ordinary therapeutic doses of radiation exert no injurious effect on it. In particular, the animal experiments indicated that, to produce direct degenerative changes of the heart muscle, extremely high doses, surpassing many times those of clinical practicability, were necessary. Changes were at times obtained from smaller doses but they were thought to be secondary to inflammatory reaction induced in the lungs which are considerably more susceptible to irradiation damage than the heart or to represent the effect of the circulation through the organ of toxic products from more generalized irradiation and accompanying disturbances of nutrition. In young animals (embryos) a retarding growth effect of the rapidly developing heart was noted. However, this, too, was explained on the basis of general inhibition of growth of the organism as a whole rather than a specific action of the irradiation on the heart itself.

The animal experiments as well as clinical observations further indicated that irradiation, either by the use of roentgen rays or radium, does not have a direct effect on the blood pressure, although a fall may result indirectly from a general systemic reaction or certain depressive effects on the vasomotor centers.

Since Desjardins' publication, there have been few additional contributions to the literature. It appears that the conclusions reached represented the final answer to the problem and that nothing worth while could be gained from continuation of the investigations.

During the last few years, however, the ever increasing popularity of the fractionated protracted method of irradiation with very large cumulative doses and the gradual extension of high voltage roentgen therapy to the supervoltage and megavoltage fields have created new clinical situations which make the opening of the subject very desirable both from the experimental and the clinical standpoint. It is fortunate, therefore, that Leach and his coworkers have undertaken the first step in this direction and that in several subsequent publications, the series of which is not yet complete, they have brought to light valuable new information which cannot fail to enrich the clinical knowledge of the radiotherapist.

The initial publications of the series deal with animal experiments. This study was made with the purpose of determining the immediate and late effect of roentgen rays on the heart of laboratory animals. As indicated in the series of Desjardins, there have been several authors who have investigated the immediate effect, but there is apparently only one published record, that of Domagk[2] on the late effect. On the other hand, clinical experience shows that in other structures or organs, such as skin, subcutaneous tissue, muscle, bone, and so forth, late damage often results when the immediate effect following the irradiation

[1] Desjardins, A. U. Action of roentgen rays and radium on the heart and lungs. AM. J. ROENTGENOL. & RAD. THERAPY, 1932, *27*, 149; 303; 477.

[2] Domagk, G. Gewebsveränderungen nach Röntgenbestrahlungen. *Ergebn. d. inn. Med. u. Kinderh.*, 1928, *33*, 1–62.

pleuropulmonitis, according to which the abnormal function of the thoracic wall, mediastinum and diaphragm is almost as important in causing the pulmonary dysfunction as the pleuropulmonary fibrosis itself. Furthermore, there is evidence accumulating that the cause of the pericarditis and adhesions between the pericardium and pleura in such instances is not a primary roentgen effect, but extension of an inflammatory reaction to those membranes from the contiguous lung. These observations are important as we shall see from the point of view of correct electrocardiographic interpretations.

In two very recent papers,[8,9] Leach finally presented the results of his painstaking clinical observations on the effect of the roentgen rays on the heart of humans. For this study 85 patients were chosen and in order to obtain comparable data they were placed in three groups. Group I included those with cancer of the head, neck, female genital tract and rectum, in which no radiation therapy was delivered to the thorax. Group II included those with malignant lymphoma or teratoma in which the irradiation was rather general, always extending to the thorax. Group III included those with cancer of the breast, lung, esophagus and cardiac end of the stomach, in which roentgen therapy was confined to the thorax alone. Apart from the routine examinations made in conjunction with the cancer, the study consisted of (1) history with particular reference to the cardiovascular system, (2) a physical examination, (3) roentgenoscopic and teleroentgen examination of the chest and heart and (4) electrocardiographic examination. All examinations were made prior to roentgen therapy and at various intervals thereafter, depending on the individual findings. The follow-up period ranged from a few months, in those who died or became lost to other institutions, to over three years.

[8] Leach, J. E. Some of the effects of roentgen irradiation on the cardiovascular system. AM. J. ROENTGENOL. & RAD. THERAPY, 1943, 50, 616–628.

[9] Leach, J. E. Effect of roentgen therapy on the heart. *Arch.*

A careful analysis of all cases of the three groups revealed that there was no evidence that roentgen therapy *per se* caused injury to the heart or that it was responsible for the complications that arose. In Group III, three patients were observed who developed chronic pericarditis or pleuropericardial adhesions following heavy roentgen therapy over the chest. However, since according to the animal experiments, the pericardium can resist even larger doses of roentgen rays than the myocardium, it is probable that the changes were secondary to the chronic infection of the thoracic wall, ribs and left lung which was present in all three instances. As the infection progressed, the pericardium became invaded because of its contiguity.

Independent from the series mentioned above, Leach also observed four cases of carotid sinus syndrome occurring during the course of roentgen therapy. These cases had metastatic carcinoma of the upper cervical lymph nodes intimately associated with the carotid bifurcation plus considerable secondary infection. The episodes consisted of sudden syncope, slow pulse, lowered blood pressure and in two cases brief epileptiform seizures which were promptly relieved by atropine or ephedrine sulfate. Following completion of the irradiation and clearing up of the infection, the episodes completely and permanently ceased. In several cases of the series, a depression of the blood pressure was noted but this was thought to be more likely due to insufficient nourishment, anemia, fever, neoplastic toxemia and only to a lesser extent to a general irradiation effect. Contrary to other observers, the author found cardiac arrhythmia developing only infrequently as a result of roentgen therapy. Whenever it occurred, it could easily be traced to some other cause.

The most outstanding findings—the ones which in fact could create a great deal of confusion—were in connection with the electrocardiographic examinations. In general, two types of variations from the normal were noted. The first occurred in pa-

tients of all the groups, quite irrespective of whether or not heart disease was present prior to the roentgen treatment. It consisted in a minor variation of all the waves, reflecting a change in the physiologic state of the myocardium. The cause may be attributed to anemia, infection, cachexia, toxemia and perhaps a depressing effect of the roentgen irradiation itself. The second alteration occurred only in patients who had radiation therapy over the thorax. It was limited to the ventricular complex and while there were frequent unpredictable changes in the voltages of the QRS waves, the variation in the configuration of the T wave seemed to follow a definite pattern. When there was an appreciable degree of irradiation pleuropneumonitis with attendant mediastinal shift and elevation of the diaphragm in the left hemithorax, the T wave in lead I became progressively lowered and even negative, while its reciprocal, T_3, became higher. Conversely, when the above changes occurred in the right hemithorax, T_1 became higher and T_3 lower, although the variations were never as marked as on the left side. Finally, when the mediastinum received the bulk of the irradiation, the alteration was the least marked, consisting of only minor variations of the voltages of the different waves. The configuration of an electrocardiogram depends, among other things, on the direction of the pathways conducting the action current from the epicardial surface of the heart to the extremities. Leach therefore assumes that the above T wave changes may well be explained on the basis of the altered relation of the heart to the thoracic wall, the elevated diaphragm and the posterior muscle mass through which the bulk of the action current is carried away from the heart. For the same reason, the alteration of the T wave appeared greatest when the irradiation was made on the left side, less marked when the irradiation was made on the right side and practically undisturbed when the irradiation was made over the mediastinum. That these electrocardiographic changes cannot be caused by injury to the heart itself Leach seems quite certain.

Obviously, it will remain for the future to furnish additional data on this very important subject. For the present, we can safely make the premise that no damage is being done to the heart itself by the various methods of irradiation as currently used in clinical roentgen therapy. The observations should be extended, however, to cover the entire life span of the treated individual and they should also include the higher ranges of supervoltage and megavoltage roentgen therapy.

T. Leucutia

SOCIETY PROCEEDINGS, CORRESPONDENCE AND NEWS ITEMS

Items for this section solicited promptly after the events to which they refer.

MEETINGS OF ROENTGEN SOCIETIES*

United States of America

American Roentgen Ray Society
Secretary, Dr. H. Dabney Kerr, University Hospital, Iowa City, Iowa. Annual meeting: Joint Meeting of American Roentgen Ray Society and Radiological Society of North America, Palmer House, Chicago, Ill., Sept. 24–29, 1944.

American College of Radiology
Secretary, Mac F. Cahal, 540 N. Michigan Ave., Chicago, Ill.

Section on Radiology, American Medical Association
Secretary, Dr. J. T. Murphy, 421 Michigan St., Toledo, Ohio. Annual meeting: Chicago, Ill., June 12–16, 1944.

Arkansas Radiological Society
Secretary, Dr. J. S. Wilson, Mack Wilson Hospital, Monticello, Ark. Meets every three months and also at time and place of State Medical Association.

Radiological Society of North America
Secretary, Dr. D. S. Childs, 607 Medical Arts Bldg., Syracuse, N. Y. Annual meeting: Joint Meeting of American Roentgen Ray Society and Radiological Society of North America, Palmer House, Chicago, Ill., Sept. 24–29, 1944.

Radiological Section, Baltimore Medical Society
Secretary, Dr. Walter L. Kilby, Baltimore. Meets third Tuesday each month, September to May.

Section on Radiology, California Medical Association
Secretary, Dr. Earl R. Miller, University of California Hospital, San Francisco, Calif.

Radiological Section, Connecticut Medical Society
Secretary, Dr. Max Climan, 242 Trumbull St., Hartford, Conn. Meets bi-monthly on second Thursday, at place selected by Secretary. Annual meeting in May.

Section on Radiology, Illinois State Medical Society
Secretary, Dr. H. W. Ackemann, 321 W. State St., Rockford, Ill.

Radiological Section, Los Angeles County Medical Association
Secretary, Dr. Roy W. Johnson, 1407 S. Hope St., Los Angeles, Calif. Meets on second Wednesday of each month at the County Society Building.

Radiological Section, Southern Medical Association
Secretary, Dr. Roy G. Giles, Temple, Texas.

Brooklyn Roentgen Ray Society
Secretary, Dr. Leo Harrington, 880 Ocean Ave., Brooklyn, N.Y. Meets monthly on fourth Tuesday, October to April.

Buffalo Radiological Society
Secretary, Dr. Joseph S. Gian-Francheschi, 610 Niagara St., Buffalo, N. Y. Meets second Monday of each month except during summer months.

Chicago Roentgen Society
Secretary, Dr. Warren W. Furey, 6844 S. Oglesby Ave. Chicago, Ill. Meets second Thursday of each month October to April inclusive at the Palmer House.

Cincinnati Radiological Society
Secretary, Dr. Samuel Brown, 707 Race St., Cincinnati, Ohio. Meets third Tuesday of each month, October to May, inclusive.

Cleveland Radiological Society
Secretary, Dr. D. D. Brannan, 11311 Shaker Blvd., Cleveland 4, Ohio. Meets at 6:30 P.M. at Allerton Hotel on fourth Monday each month, October to April, inclusive.

Dallas-Fort Worth Roentgen Study Club
Secretary, Dr. X. R. Hyde, Medical Arts Bldg., Fort Worth, Texas. Meetings held in Dallas on odd months and in Fort Worth on even months, on third Monday, at 7:30 P.M.

Denver Radiological Club
Secretary, Dr. Edward J. Meister, 966 Metropolitan Bldg. Denver, Colo. Meets third Friday of each month at Denver Athletic Club.

Detroit Roentgen Ray and Radium Society
Secretary, Dr. E. R. Witwer, Harper Hospital. Meets monthly on first Thursday from October to May, at Wayne County Medical Society Building.

Florida Radiological Society
Acting Secretary, Dr. Walter A. Weed, 204 Exchange Bldg. Orlando, Fla. Meetings in May and November.

Georgia Radiological Society
Secretary, Dr. James J. Clark, 478 Peachtree St., Atlanta Ga. Meets in November and at annual meeting of Medical Association of Georgia in the spring.

Radiological Society of Kansas City
Secretary, Dr. Arthur B. Smith, 800 Argyle Bldg., Kansas City, Mo. Meets third Thursday of each month at a place designated by the president.

Illinois Radiological Society
Secretary, Dr. Wm. DeHollander, St. John's Hospital Springfield, Ill. Meets three times a year.

Indiana Roentgen Society
Secretary, Dr. H. C. Ochsner, Methodist Hospital, Indianapolis. Meeting held the second Sunday in May annually.

Kentucky Radiological Society
Secretary, Dr. W. C. Martin, 321 W. Broadway Louisville Meets annually in Louisville on first Saturday in April.

Long Island Radiological Society
Secretary, Dr. Marcus Wiener, 1430-48th St., Brooklyn, N. Y. Meets Kings County Med. Soc. Bldg. monthly on fourth Thursday, October to May, 8:30 P.M.

Louisiana Radiological Society
Secretary, Dr. J. R. Anderson, 1130 Louisiana Ave., Shreveport. Meets annually during Louisiana State Medical Society Meeting.

Michigan Association of Roentgenologists
Secretary, Dr. E. M. Shebesta, 1429 David Whitney Bldg., Detroit. Three meetings a year, Fall, Winter, Spring.

Milwaukee Roentgen Ray Society
Secretary, Dr. C. A. H. Fortier, 231 W. Wisconsin Ave., Milwaukee, Wis. Meets monthly on second Monday at University Club.

Minnesota Radiological Society
Secretary, Dr. Annette T. Stenstrom, 1218 Medical Arts Bldg., Minneapolis, Minn. One meeting a year at time of Minnesota State Medical Association.

Nebraska Radiological Society
Secretary, Dr. D. A. Dowell, Medical Arts Bldg. Omaha, Nebr. Meets third Wednesday of each month, at 6 P.M. at either Omaha or Lincoln.

New England Roentgen Ray Society
Secretary, Dr. Hugh F. Hare, Lahey Clinic, Boston, Mass. Meets monthly on third Friday, Boston Medical Library.

Radiological Society of New Jersey
Secretary, Dr. H. J. Perlberg, 921 Bergen Ave., Jersey City. Meets annually at time and place of State Medical Society. Mid-year meetings at place chosen by president.

New York Roentgen Society
Secretary, Dr. Ramsay Spillman, 115 East 61st St., New York City. Meets monthly on third Monday, New York Academy of Medicine, at 8:00 P.M.

North Carolina Roentgen Ray Society
Secretary, Dr. Major Fleming, Rocky Mount, N. C. Annual meeting at time and place of State Medical Society. Mid-year scientific meeting at place designated.

* Secretaries of Societies not here listed are requested to send the necessary information to the Editor.

North Dakota Radiological Society
Secretary, Dr. L. A. Nash, St. John's Hospital, Fargo. Meetings held by announcement.

Central New York Roentgen Ray Society
Secretary, Dr. C. F. Potter, 820 S. Crouse Ave., Syracuse. Three meetings a year. January, May, November.

Ohio Radiological Society
Secretary, Dr. J. E. McCarthy, 707 Race St., Cincinnati. Meets at time and place of annual meeting of Ohio State Medical Association.

Pacific Roentgen Society
Secretary, Dr. L. H. Garland, 450 Sutter St., San Francisco, Calif. Meets annually, during meeting of California Medical Association.

Pennsylvania Radiological Society
Secretary, Dr. L. E. Wurster, 416 Pine St., Williamsport. Annual meeting at Penn Alto Hotel, Altoona, Pa., May 13–14, 1944.

Philadelphia Roentgen Ray Society
Secretary, Dr. R. P. Barden, University Hospital, Meetings first Thursday of each month from October to May inclusive at 8:15 P.M., in Thompson Hall, College of Physicians, 19 S. 22d St.

Pittsburgh Roentgen Society
Secretary, Dr. R. G. Alley, 4800 Friendship Ave. Meets second Wednesday each month, 4:30 P.M., October to June, Pittsburgh Academy of Medicine.

Rochester Roentgen Ray Society, Rochester, N. Y.
Secretary, Dr. Sidney Larson, Strong Memorial Hospital. Meets monthly on third Monday from October to May, inclusive, 8 P.M. at Strong Memorial Hospital.

Rocky Mountain Radiological Society
Secretary, Dr. A. M. Popma, 220 N. First St., Boise, Idaho.

St. Louis Society of Radiologists
Secretary, Dr. P. C. Schnoebelen, 462 N. Taylor Ave. Meets fourth Wednesday of each month, except June, July, August, and September, at a place designated by the president.

San Diego Roentgen Society
Secretary, Dr. Henry L. Jaffe, Naval Hospital, Balboa Park, San Diego, Calif. Meets monthly on first Wednesday at dinner.

San Francisco Radiological Society
Secretary, Dr. Martha Mottram, 450 Sutter St., San Francisco. Meets monthly on third Thursday at 7:45 P.M., first six months of year at Toland Hall, University of California Hospital, second six months at Lane Hall, Stanford University Hospital.

Shreveport Radiological Club
Secretary, Dr. R. W. Cooper, Charity Hospital, Shreveport, La. Meets monthly on third Wednesday, at 7:30 P.M., September to May inclusive.

South Carolina X-Ray Society
Secretary, Dr. T. A. Pitts, Baptist Hospital, Columbia, S. C. Meets in Charleston on first Thursday in November, also at the time and place of South Carolina State Medical Association.

Tennessee Radiological Society
Secretary, Dr. J. M. Frère, 707 Walnut St., Chattanooga, Tenn. Meets annually at the time and place of the Tennessee State Medical Association.

Texas Radiological Society
Secretary, Dr. Herman Klapproth, Sherman, Texas.

University of Michigan Department of Roentgenology Staff Meeting
Meets each Monday evening from September to June, at 7 P.M. at University Hospital.

University of Wisconsin Radiological Conference
Secretary, Dr. E. A. Pohle, 1300 University Ave., Madison, Wis. Meets every Thursday from 4:00–5:00 P.M., Room 301, Service Memorial Institute.

Virginia Radiological Society
Secretary, Dr. E. L. Flanagan, 116 E. Franklin St., Richmond, Va. Meets annually in October.

Washington State Radiological Society
Secretary, Dr. Thomas Carlile, 1115 Terry St., Seattle. Meets fourth Monday each month, October through May, College Club, Seattle.

X-ray Study Club of San Francisco
Secretary, Dr. J. M. Robinson, University of California Hospital. Meets monthly in evening on third Thursday.

Cuba

Sociedad de Radiología y Fisioterapia de Cuba
President, Dr. J. Manuel Viamonte, Hospital Mercedes, Habana, Cuba. Meets monthly in Habana.

British Empire

British Institute of Radiology Incorporated with the Röntgen Society
Medical Members' meeting held monthly on third Friday at 2:30 P.M. and Ordinary Meeting at same time on following Saturday, October to May, 32 Welbeck St., London, W.1.

Section of Radiology of the Royal Society of Medicine (Confined to Medical Members)
Meets on the third Friday of each month at 4:45 P.M. at the Royal Society of Medicine 1, Wimpole St., London, W. 1.

Faculty of Radiologists
Secretary, Dr. M. H. Jupe, 32 Welbeck St., London, W. 1 England.

Section of Radiology and Medical Electricity, Australasian Medical Congress
Secretary, Dr. H. M. Cutler, 139 Macquarie St., Sydney, New South Wales.

Radiological Section of the Victorian Branch of the British Medical Association
Secretary, Dr. Keith Hallam, St. George's Hospital, K.E.W., Melbourne, E. 4, Victoria, Australia. Meets monthly from March to November inclusive.

Canadian Association of Radiologists
Secretary, Dr. A. D. Irvine, 540 Tegler Bldg., Edmonton, Alberta.

Section of Radiology, Canadian Medical Association
Secretary, Dr. C. M. Jones, Inglis St., Ext. Halifax, N. S.

Radiological Section, New Zealand British Medical Association
Secretary, Dr. Colin Anderson, Invercargill, New Zealand. Meets annually.

South America

Sociedad Argentina de Radiologia
Secretary, Dr. Guido Gotta, Buenos Aires, Argentina. Meetings are held monthly.

Continental Europe

Sociedad Espanola de Radiologia y Electrologia
Secretary, Dr. J. Martin-Crespo, Fuencarral, 7. Madrid, Spain. Meets monthly in Madrid.

Société Suisse de Radiologie (Schweizerische Röntgen-Gesellschaft)
Secretary for French language, Dr. A. Grosjean La Chaux de Fonds.
Secretary for German language, Dr. Scheurer, Molzgasse Biel. Meets annually in different cities.

Societatea Romana de Radiologie si Electrologie
Secretary, Dr. Oscar Meller, Str. Banul Mărăcine, 30, S. I., Bucuresti, Roumania. Meets second Monday in every month with the exception of July and August.

All-Russian Roentgen Ray Association, Leningrad: USSR
the State Institute of Roentgenology and Radiology, 6 Roentgen St.
Secretaries, Drs. S. A. Reinberg and S. G. Simonson. Meets annually.

Leningrad Roentgen Ray Society
Secretaries, Drs. S. G. Simonson and G. A. Gusterin. Meets monthly, first Monday at 8 o'clock, State Institute of Roentgenology and Radiology, Leningrad.

Moscow Roentgen Ray Society
Secretaries, Drs. L. L. Holst, A. W. Ssamygin and S. T. Konobejevsky. Meets monthly, first Monday, 8 P.M.

Scandinavian Roentgen Societies
The Scandinavian roentgen societies have formed a joint association called the Northern Association for Medical Radiology, meeting every second year in the different countries belonging to the Association

Announcing . . .

A JOINT MEETING

of

THE AMERICAN ROENTGEN RAY SOCIETY

and

THE RADIOLOGICAL SOCIETY OF NORTH AMERICA

at

THE PALMER HOUSE, CHICAGO, ILLINOIS

SEPTEMBER 24 TO 29, 1944

The officers and members of the Program Committees of the American Roentgen Ray Society and the Radiological Society of North America, at a meeting held in Chicago, February 12, 1944, decided that the interests of all radiologists on the North American continent could best be served by conducting a joint meeting in 1944.

It appears obvious that, by having such a meeting, radiologists in military and civilian practice could obtain much valuable information regarding the recent technical and scientific advances in radiology. The dissemination of such knowledge is recognized as a valuable contribution to the successful prosecution of our military activities.

Because of its central location, Chicago was selected as the most desirable city for such a meeting. Prompt action was necessary in order to obtain adequate hotel facilities, and we have been assured that sufficient rooms will be made available at the Palmer House for those who wish to attend.

It is not too early to make your plans for the meeting, and you are invited to submit contributions for the scientific program and exhibits as soon as possible.

LYELL C. KINNEY, *President-Elect*
American Roentgen Ray Society

ELDWIN R. WITWER, *President*
Radiological Society of North America

DEPARTMENT OF TECHNIQUE

Department Editor: Robert B. Taft, M.D., B.S., M.A., 103 Rutledge Ave. Charleston, S. C.

A RADIOPAQUE LIQUID LATEX INJECTION MEDIUM FOR BLOOD VESSELS

By C. E. TOBIN
Department of Anatomy, The University of Rochester School of Medicine and Dentistry
ROCHESTER, NEW YORK

A postmortem study of main or collateral blood vessels is desired frequently in conditions where disease or surgical intervention have occluded parts of the vascular system. In such studies, an injection medium which possesses radiopaque properties, in addition to filling the desired vessels with a pigmented rubber-like medium, would be the ideal solution whereby the course of the vessels involved could be determined by roentgenograms and by subsequent dissection.

Pigmented liquid latex, an aqueous, colloidal, rubber solution, has been found to be an ideal injection medium for dissection of blood vessels in laboratory specimens because of its ability to: (1) be colored with fast, soluble dyes; (2) penetrate into the smallest vessels without staining adjacent tissue; (3) be used cold and be diluted to the desired consistency by addition of distilled H_2O; (4) set into a tough, flexible cast of vessels (where not deposited in too large masses) by reacting with phenol, phenolic compounds, formalin or acids.[1] A syringe with a glass cylinder and rubber piston should be used for injection, as latex will cause the plunger of "all glass" or "all metal" syringes to adhere to the cylinder walls.

Pigmented latex alone has very little radiopacity even when "soft tissue technique" is used. The following experiments were tried, therefore, to find a suitable radiopaque medium which could be mixed with pigmented latex and still retain the qualities of latex listed above.

Samples of red liquid latex (pH 9.9) were mixed with sodium iodomethane sulfonate (skiodan), crystalline potassium iodide, barium sulfate, tetraiodophenolphthalein sodium salt (tetradol emulsion), and ethyl iodophenylundecylate (pantopaque) and tested by injection into rats.* Each of the first four substances when added directly to the stock solution of latex coagulated it, making these combinations unsuitable for injection. However, a small quantity of these substances could be added to the latex, but the amount was insufficient to produce a continuous roentgenographic shadow in the injected animals, even though the mixture remained fluid for injection at pH 10 (Fig. 1). Pantopaque, on the other hand, was found to form a stable emulsion with latex, but 50 per cent or more of this substance had to be mixed with the latex for good contrast (Fig. 2 and 3). Pantopaque, being an oily medium, caused an increase in the viscosity of the latex-pantopaque mixture, which could be overcome partly by raising the pH to 10.78 with the addition of NH_4OH. The arteries of the material studied were injected as soon as possible after death with pantopaque-latex mixture, the specimens roentgenographed after injection, and later preserved either with our stock of embalming solution (pH 2.6) or with a

[1] Gamble, D. L. Liquid latex as an injection mass for blood-vessels. *Science*, 1939, 90, 520.

* The radiopaque media were furnished by the Department of Radiology, University of Rochester School of Medicine and Dentistry.

formalin-acetic acid mixture (pH 3.3). Several days later when the latex had hardened, the specimens were dissected to confirm the extent of the injection.

The clinical value of this injection medium, in studying the vascular system, is demonstrated by the following case, which will be presented more in detail as a separate report from the Department of Surgery, The University of Rochester School of Medicine and Dentistry.

J. G., white male, aged fifty-two. Dissecting

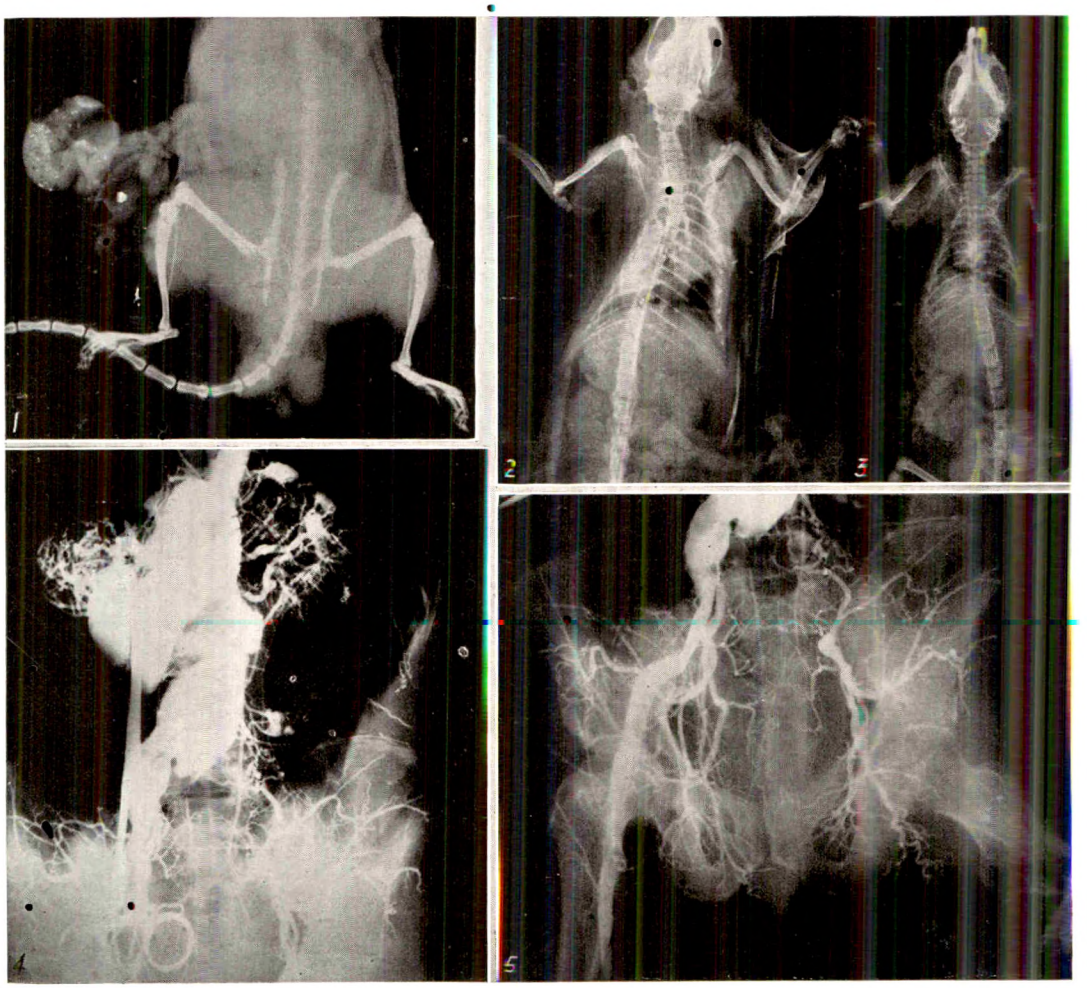


Fig. 1. Discontinuous shadow made by a saturated solution of potassium iodide-latex in the viscera of a rat. Similar shadows were made by a saturated solution of barium, skiodan and tetradol emulsion when mixed with latex. The arteries of this and the other experimental animals were filled with the injection medium.

Fig. 2. A continuous outline of the right carotid artery of a rat was seen after injection with 50 per cent pantopaque-latex.

Fig. 3. A 20 per cent pantopaque-latex mixture was insufficient to produce a distinct shadow.

Fig. 4. Roentgenogram of case reported showing shadows from the main and smaller arteries as well as the aortic and right iliac aneurysm, and portion of the jejunum (at tip of clamp) filled with injection medium.

Fig. 5. Roentgenogram of case reported showing aneurysm of aorta and right iliac artery, as well as all the branches of the right iliac and hypogastric artery, filled with the contrast medium. The left iliac artery is obliterated but branches of the lumbar and sacral arteries formed anastomotic channels with the left hypogastric. The branches of the latter artery, although enlarged, were patent and normal in their distribution to the gluteal region and left stump. No enlargement of the hemorrhoidal vessels is seen.

aneurysm and thrombosis of left common iliac artery in addition to aneurysmal dilatation both of aorta, proximal to its bifurcation, and of proximal part of right iliac artery, were ascertained by laparotomy on January 25, 1942. The aorta was occluded by two tape ligatures between the origin of the inferior mesenteric artery and the aortic aneurysm, January 29. The left extremity was amputated at mid-thigh on February 9. The left stump and right extremity showed no subsequent signs of necrosis and sufficient collateral channels were assumed to have been established. To relieve pain from the "phantom left limb," lumbar block and division of sciatic trunks were performed on June 16. Subsequently the patient showed a steady decline with continuous loss of blood from the gastrointestinal tract via rectum.

The thorax was opened within two hours after death (August 28, 1942) and approximately 1 quart of pantopaque-latex injected by means of a Janet-Frank's bladder syringe connected to a cannula in the descending thoracic aorta. The pigmented injection medium filled all the arteries to the gastrointestinal tract and some of the medium was observed to enter the lumen of the proximal part of the jejunum. The right posterior tibial artery was exposed and found to contain pigmented injection medium, verifying the completeness of the injection. The liver, stomach, small (except duodenum and upper part of jejunum) and large intestine were removed. Roentgenograms of the entire specimen were taken and the specimen fixed in acetic acid embalming solution (pH 2.6) by immersion and "punch-embalming" for later dissection.

At the time of death the vessels involved in supplying the right extremity and the cause of loss of blood from the gastrointestinal tract (via rectum) were not ascertained. It was tentatively assumed that collateral circulation through dilated hemorrhoidal vessels might play a part, if the right iliac vessels were occluded. Roentgenograms showed, however, that the proximal part of the jejunum (Fig. 4—at point of clamp), the aneurysm of both the aorta and right iliac artery and all the pelvic branches of the latter, as well as arteries of the right extremity were filled with the contrast medium. The left common iliac artery was completely occluded, but the left hypogastric artery and its branches were patent (Fig. 5). No dilatation of the hemorrhoidal vessels could be observed.

Subsequent dissection demonstrated that the ligatures had cut through the aortic walls and were embedded in an aneurysm, which developed after ligation, proximal to the origin of the inferior mesenteric artery. This second aneurysm had extended over the point of ligation and produced a new channel through which blood could flow distally. A perforation into the jejunum, near the duodenojejunal junction, was found which formed a direct communication from the aneurysm, above the inferior mesenteric, into the gastrointestinal tract.

As indicated in these figures and verified by dissection, the vessels to the right limb had patent lumina, as shown by their containing red latex, whereas lumbar and sacral arteries of the left side formed collateral channels to the left hypogastric artery, the branches of which were slightly enlarged but normal in their distribution. Even the smaller arteries, such as the lateral sacral, were filled with the latex mixture and their course and anastomosis could be dissected with ease.

Although in the present emergency there may be a scarcity of latex, biological supply houses or other depots may have sufficient stores to warrant the use of this injection medium for studies as described.

SUMMARY

Ethyliodophenylundecylate (pantopaque) emulsified with pigmented liquid latex is an ideal injection medium for studies of the vascular system where the course of main or collateral vessels has been occluded by disease or surgical intervention. Roentgenograms of the vessels can be obtained and their course ascertained by dissection.

• I am greatly indebted to Dr. J. J. Morton and Dr. W. J. M. Scott of the Department of Surgery and Drs. S. L. Warren and W. H. Strain of the Department of Radiology for making the material available and for their cooperation in this study.

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ABSTRACTS OF ROENTGEN AND RADIUM LITERATURE

ROENTGEN DIAGNOSIS

NECK AND CHEST

Douglas, Bruce H., and Birkelo, Carl C. Screening for tuberculosis in a civilian population by fluorography. *Ann. Int. Med.*, Nov., 1941, *15*, 853–857.

A rather common procedure for survey of pulmonary tuberculosis includes tuberculin skin testing to identify those who at some time may have been infected with the tubercle bacillus; thereafter, roentgenographically examining the chests of all positive skin reactors.

The authors have inventoried other possibilities which might provide for more direct information and money savings. They have considered the full-size standard film but have concluded it to be too expensive; they have considered sensitized paper—less costly but still too expensive; they object to fluoroscopy because of its not providing a permanent record and because of Fellows' statistics which have indicated approximately a 13 per cent error for it versus the standard film; they believe that the 35 millimeter film sacrifices too much accuracy in favor of economy. They have concluded that a 4×5 inch fluorograph is reasonably accurate, the single image study incurring no more than 2.6 per cent error as compared with the standard 14×17 inch film studies. They recommend this procedure as practical for group surveys and substantiate their contentions on the basis of 30,000 examinations. Some of these have been studied by skin testing also. Of 47.6 per cent who reacted positively, there were 3 active cases; whereas, among 671 who reacted negatively, there were 2 active cases. A number of those who were inoculated with tuberculin failed to return for re-examination.—*A. A. de Lorimier.*

Bailey, Wilbur. Technic for chest x-ray examinations of large groups. I. The use of standard size films. *Radiology*, Sept., 1942, *39*, 306–309.

Roentgen examination of large groups of people for tuberculosis is becoming increasingly popular and of course is used for the examination of Army recruits. Films of substandard size and film substitutes have been used in an attempt to reduce the cost of such examinations and the impression has been given in some quarters that satisfactory results and sufficient speed could not be attained with standard 14×17 inch films. The author maintains that this is not true and describes the technique of examination used at the Los Angeles Army Induction Station. The greater part of the time required for any mass survey is consumed in measuring and placing the patients and therefore this method can be made as rapid as any of those that use substandard films, paper rolls, etc., while the films are of much better quality.

Reports in the German literature say that 300 men can be examined per hour but the author has found that for good work not more than 100 can be examined as a matter of routine. More than 125 per hour were examined when the subjects were medical students, as they gave more intelligent cooperation.—*Audrey G. Morgan.*

Bailey, Wilbur. Technic for chest x-ray examinations of large groups. II. The use of miniature films. *Radiology*, Sept., 1942, *39*, 310–313.

The technique used for mass examinations with 4×5 and 4×10 inch films does not differ greatly from that with standard films. The target-film distance is decreased, however, and the kilovoltage increased, while the milliampere-second output must rise to 40 to 60 ma-sec., or even more. These changes tend to produce poorer detail and contrast in the image. But though the detail is less than on standard films a comparison of a large number of cases examined with standard films and an equally large number with 4×10 inch films showed that a somewhat larger number of cases of tuberculosis was revealed by the 4×10 inch films than by the standard films.

Another disadvantage of the small films lies in the fact that the long exposure time required is apt to produce a simulated enlargement of the heart and great vessels and it is hard to tell whether there is an actual enlargement. But as

a whole the results are better than might be expected for mass examinations for tuberculosis. —*Audrey G. Morgan.*

KERR, WM. J. Faulty movements of the diaphragm as a cause of non-obstructive emphysema and angina pectoris. *Radiology*, August, 1942, *39*, 153–156.

The author maintains that non-obstructive emphysema and apparent angina pectoris are often brought on by faulty movements of the diaphragm due to obesity. He has very successfully treated such cases of chronic bronchitis with emphysema and relieved the orthostatic dyspnea and cough by the use of an abdominal belt which is illustrated. This supporting belt acts as a means of artificial respiration and helps to restore the function of the diaphragm. Kountz and Alexander recommended the use of a firm supporting belt to hold up the pendulous abdomen and aid in the movements of the diaphragm. The author modified this belt by adding elastic segments at the sides to insure freer movement of the diaphragm.—*Audrey G. Morgan.*

ALLISON, STANTON T. The vanishing lung; report of a case of advanced bullous emphysema. *Ann. Int. Med.*, July, 1942, *17*, 139–148.

Pulmonary emphysema is a condition of the lungs brought about by overdistention of the walls of the alveoli resulting in loss of elasticity and finally rupture. This may produce such pressure on the lung tissue that it is almost entirely destroyed.

A case is described in a man of forty-one who complained of a severe chest cold with pain and dyspnea. He had had epilepsy since childhood with infrequent attacks of petit mal. He had had pneumonia twice before he was ten years of age. While in college in 1917 his chest expansion had been 4 inches; in 1940 on taking an examination for conditioning he was found to have no chest expansion at all but in spite of that was allowed to take the strenuous conditioning exercises, after which he suffered from severe and long-continued dyspnea. For some months he had been unable to sleep on his left side without cough and dyspnea.

Stereoscopic roentgenograms of the chest showed no lung markings in the right chest except a few cobweb-like strands at the base. The upper half of the left lung showed the brilliance characteristic of absence of lung tissue but the markings of the lower half of the left field were increased. When he slept on his left side he splinted the only part of the lung that was functioning and so of course became dyspneic.

This patient had had frequent respiratory infections during childhood and had had what he called a cigarette cough for years. Emphysema is caused by strain on the walls of the alveoli due to coughing in patients with asthma, chronic bronchitis and other chronic pulmonary diseases. The popular theory that it is caused by glass blowing or the blowing of wind instruments is false.

The patient was advised to go to Tucson, Arizona, where the clear, dry air will lessen the danger of respiratory infections, to avoid strenuous exercise and heavy lifting and be at least very moderate in the use of tobacco and alcohol, as the former would further irritate the bronchi and the latter would increase the pulse rate and lower his resistance. A diet low in fats and carbohydrates but high in proteins, vegetables, fruits and vitamins was recommended with the use of an abdominal belt to increase intra-abdominal pressure, elevate the diaphragm and increase vital capacity. If these directions are followed it is believed he may live for years even with his limited lung capacity.—*Audrey G. Morgan.*

KARAN, A. A., and SINGER, EMANUEL. Transitory pulmonary infiltrations mistaken for tuberculosis, with a report of five cases. *Ann. Int. Med.*, July, 1942, *17*, 106–124.

Recently increasing dependence has been placed on the diagnosis of pulmonary tuberculosis by roentgen examination. Five cases are described which show that roentgenograms of themselves are not entirely reliable, particularly a single examination. A series of examinations should always be made. If tubercle bacilli cannot be found in the sputum or stomach contents by microscopic examination, cultures or guinea pig inoculation, the case is probably not one of tuberculosis. This is important as artificial pneumothorax, which is useful in the treatment of tuberculosis, may be useless or even harmful in other conditions.

The roentgenograms in the 5 cases described which were accepted as typical of tuberculosis by competent roentgenologists and phthisiologists, are reproduced. But serial examinations showed the incorrectness of the diagnosis as the lung infiltrations cleared up.

Two of the cases were bronchopneumonia probably caused by a filtrable virus. In 1 case the infiltration and rarefaction seen on the first roentgenogram were thought to be due to a lobular atelectasis with beginning necrosis of the lung resulting from bronchial obstruction. The other 2 cases showed eosinophilia and were probably allergic in character. The agent that caused the allergic reaction was not determined. In 1 of the cases the pulmonary exudate had persisted long enough to cause increased resistance in the pulmonary circulation, resulting in enlargement of the right ventricle which returned to normal size when the exudate was absorbed.—*Audrey G. Morgan.*

Reeves, Robert J., Smith, David T., and Baker, Roger D. [Tuberculous pneumonia of lower lobe of right lung (primary focus).] *Radiology*, Oct., 1942, *39*, 467–473.

The case considered at this clinico-pathological conference was that of a white grocery salesman admitted to the hospital because of fever of six weeks' duration. Unusual features of the case were that he had a fever four months before admission that was mistaken for malaria. Six weeks before admission he had an acute respiratory infection with yellow sputum that was reported as foul. Four days before admission he had red swollen ankles with erythema. There was a persistent neutropenia with nonsegmented polymorphonuclear cells, suggesting leukemia, nodes in the right hilar region, a spreading pulmonary lesion, an enlarged spleen, a continuous high fever (39° to 40° C.) with bradycardia followed by tachycardia and a terminal gangrene of the cheek. The patient died sixty-nine days after admission to hospital.

The possible diagnoses to be considered were tuberculosis, metastatic carcinoma from some unknown origin, primary bronchogenic carcinoma, Hodgkin's disease, aleukemic leukemia, myelogenous leukemia. Autopsy showed tuberculosis, but not the usual type of adult tuberculosis with predominant involvement of the apices with nodules or cavities. It rather resembled the primary tuberculosis of childhood. Apparently the patient became infected with tuberculosis only a few months before death. The caseous pneumonia of the right lower lobe represented the primary focus, this spread to the hilar nodes causing the mass. Hematogenous spread occured to an extent that justified the term miliary tuberculosis. The fusospirochetal gangrene of the right cheek was a terminal incident not connected with the primary disease.—*Audrey G. Morgan.*

Perrone, J. A., and Levinson, J. P. Primary carcinoma of the lung (report of 115 cases, 38 autopsies and 77 bronchoscopic biopsies). *Ann. Int. Med.*, July, 1942, *17*, 12–25.

The authors present a study of 38 cases of primary carcinoma of the lung found on autopsy and 77 cases diagnosed by bronchoscopic examination at Mercy Hospital, Pittsburgh, from 1911 to 1939.

Many forms of chronic irritation of the lung seem to cause cancer. There are about 3 cases in males to 1 in females and the proportion of women has not increased in recent years in spite of the fact that women smoke a great deal more. The total incidence of carcinoma of the lung has increased quite rapidly since the beginning of the century. The age of the patients in this series was from twenty-three to seventy-six. These tumors seem to be relatively rarer in the Negro than in the white race.

About 70 per cent of the cases originate in the bronchi, and bronchoscopy is the one certain method of diagnosis. The physical findings may simulate those of any kind of lung disease. The chief roentgen finding is atelectasis of the lung. There was superimposed inflammation in about 27 per cent of these cases. Diagnosis cannot be made by roentgen examination alone.

The four cardinal symptoms are cough, hemoptysis, pain and dyspnea. Every family physician should know that in any case of indefinite disease of the lung a most thorough examination should be made for carcinoma. If bronchoscopic and bioscopic examinations were made in all such cases these tumors would be detected much earlier when the prospects for successful treatment are better.

Only 30 of the 77 cases examined in this series were operable and only 1 of them is alive three years after pneumonectomy. Surgery, however, is the treatment of choice as neither roentgen irradiation nor implantation of radium is very effective.—*Audrey G. Morgan.*

Ochsner, Alton, and DeBakey, Michael. Carcinoma of the lung. *Arch. Surg.*, Feb., 1941, *42*, 209–258.

Recently early diagnosis and improved surgical management have rendered the prognosis in carcinoma of the lung relatively favorable as

compared with earlier cases, diagnosed late and treated with inadequately developed preoperative, anesthetic and operative techniques. This disease has become a significant problem in cancer therapy; its incidence is increasing, absolutely, to be second only to cancer of the stomach (autopsy series).

Among the various suggested etiologic agents; insufficient evidence exists to incriminate influenza, tuberculosis, syphilis and inhalation of tar and benzene products.

The importance of nonspecific pulmonary infections, bronchiectasis and chronic bronchitis, pneumoconiosis and trauma is doubtful. Smoking, especially cigarette smoking associated with inhaling, is largely accountable for the increased incidence of pulmonary carcinoma in the authors' opinion.

Three-fourths of the cases are in males; four-fifths are between the ages of forty and seventy, but cases occur as early as the first decade.

Carcinoma of the lung is bronchogenic in origin, arising centrally in most instances. A logical classification of these neoplasms is Halpert's based on the development of the lining cells of the bronchus. According to this explanation, carcinomas arise by atypical proliferation from "reserve cells" (normally found beneath and developing into the ciliated cylindrical and goblet cells) to result in (1) "reserve cell," (2) cylindrical cell, or (3) squamous cell carcinoma. Metastasis occurs by direct extension, through lymphatics, through blood stream and by "bronchial embolism." Metastatic sites, in order of frequency are: regional lymph nodes (75.9 per cent), liver (33.4 per cent), bone (20.4 per cent), adrenals, kidney, brain, heart and pericardium, and pancreas. The need for removal of regional nodes at the time of operation is evident.

Bronchial carcinoma presents no characteristic symptomatology; the onset is usually insidious; the most constant symptom is cough, non-productive at first, later becoming productive of mucoid, mucopurulent, blood tinged (hemoptysis is infrequent, however) or even foul sputum. Symptoms of an acute respiratory infection or indefinite thoracic pain are fairly frequent first symptoms; dyspnea is a late complaint, as are weight loss, weakness, cyanosis and pulmonary osteoarthropathy. Peripherally located tumors may produce evidences of pleurisy with effusion. Carcinoma of the lung must be seriously considered in any patient past the age of forty who exhibits respiratory symptoms; lung abscess without apparent cause should be counted of malignant origin until proved otherwise. The interval between onset of symptoms and death is usually cited in terms of months, but such an apparently rapid course is not necessarily an indication of the true duration of the symptoms. Physical findings are also varied; obvious physical signs usually signify inoperability.

A high index of suspicion is necessary for the early diagnosis of bronchial carcinoma. "Whereas roentgenographic examination without the use of contrast mediums is usually of little or no value in the *early* diagnosis of non-obstructive bronchial neoplasms; careful stereo-roentgenographic studies are necessary for all such lesions." The roentgen interpretation of centrally located lesions is generally more difficult because of the confusion with hilar shadows produced by other lesions and by normal structures. This is particularly significant because most pulmonary neoplasms occur in the hilar region." Later, atelectasis develops due to bronchial obstruction by the carcinoma. The high incidence of correct diagnosis by roentgen examination indicates the value of this procedure; tomographic studies are reported of value. Bronchoscopy and biopsy yield a positive diagnosis in over 70 per cent of the cases. Bronchography is an additional, important means of diagnosis. Malignant cells can be recognized in sputum or pleural fluid by a pathologist trained in such methods. Aspiration biopsy carries with it the probability of spreading metastases to the pleura.

The only curative treatment of pulmonary carcinoma is total surgical extirpation of the involved lung; excision of regional nodes is necessary. Irradiation "is of value in hopeless cases in alleviating the manifestations." Ten days to two weeks preliminary to pneumonectomy artificial pneumothorax is instituted to compress the pulmonary capillary bed gradually and allow complete compensation of the cardiovascular system before operation; this procedure also permits the determination of the presence and extent of adhesions.

The authors state their preference for cyclopropane inhalation anesthesia under positive pressure, an anterior approach and individual isolation of hilar structures. In 94 collected cases 38 patients recovered and 56 (59.5 per cent) died; of 24 patients in whom massive

ligation of hilar structures was done, 5 patients recovered from the operation and 19 (79.1 per cent) died; of 54 patients in whom individual ligation of hilar structures was done, 25 recovered and 29 (53.7 per cent) died.—*Henry G. Moehring.*

Scott, Wendell G., and Lionberger, John R., Jr. Studies of the pulmonary vessels by means of body-section radiography. *Radiology*, August, 1942, *39*, 157–165.

The pulmonary vessels can be visualized better by means of body section roentgenography than by conventional roentgenography because the former method blurs out the unwanted shadows and leaves only the structures that are of interest in clear relief.

Kieffer and Moore's laminagraph was used in making a series of body section roentgenograms which are reproduced. First a series of conventional films was made in the postero-anterior, right and left anterior oblique and left lateral positions. By analyzing the location of the pulmonary vessels on these films it was possible to determine the approximate levels in the various positions in which the sectional films were to be made. This provides a set of conventional films and one of sectional films of the pulmonary vessels for comparison. The spiral method was used to get maximum blurring of unwanted shadows.

The size and degree of dilatation of the pulmonary vessels, particularly the arteries, are best shown in lateral sectional films. Dilatation is the only reliable objective sign of disease of the pulmonary arteries. It is primarily the pulmonary veins that are dilated, the arteries only slightly so, in patients with mitral stenosis and insufficiency from rheumatic heart disease.

There is a great deal of variation in the normal size, shape, position and course of the pulmonary vessels. The pulmonary veins are harder to visualize than the arteries because they are smaller, have shorter main trunks and are therefore in closer contact with the heart.

It is hoped that this method of body section roentgenography will stimulate closer study and observation of the pulmonary circulation, resulting in a deeper knowledge of its diseases. —*Audrey G. Morgan.*

Stanford, W. R., and Nalle, Brodie C., Jr. Some notes on cystic disease of the lungs, with a report of one case. *Ann. Int. Med.*, July, 1942, *17*, 65–77.

A case of cystic disease of the lungs seen in Duke Hospital is described and illustrated with photographs of the autopsy specimens and roentgenograms. The chief emphasis is laid on the difficulty of differential diagnosis between congenital cysts, acquired cysts and extensive emphysema with formation of bullae. The differentiation is important as some forms of cyst of the lung can be treated surgically.

The patient was a Negro of thirty-four who complained of shortness of breath and indigestion which had begun about four years before. He had slight wheezing at times and one physician had diagnosed the condition as asthma. These cases may present a marked resemblance to asthma.

Roentgen examination showed cysts in the left mid lung and throughout the right lung. The picture resembled that of congenital cystic disease of the lungs. An attempt was made to do a diagnostic pneumothorax on the right chest but unfortunately a spontaneous pneumothorax developed, the patient became much more dyspneic and died five days later.

The pathologist reported that this resembled a case of acquired pulmonary emphysema. There is nothing in the history to indicate that the patient had any pulmonary malformation before the age of thirty. The most likely explanation seems to be that the elastic tissue of the lung was poor in quality and that at the age of thirty it had deteriorated so much that this extensive emphysema developed.

Lack of pigment in the wall of a cyst is the only thing to show that it is congenital. But in this case there was no fluid in the cysts, they all contained air and therefore naturally pigment. An absolute diagnosis of congenital cyst cannot be made without roentgen evidence of such a lesion at birth.

The mortality in these cysts is high. Infection is a serious complication. Of course if too much lung tissue is destroyed no medical or surgical treatment will do any good.—*Audrey G. Morgan.*

Winn, William A., and Johnson Gilbert H. Primary coccidioidomycosis; a roentgenographic study of 40 cases. *Ann. Int. Med.*, Sept., 1942, *17*, 407–422.

Human infection with *Coccidioides immitis* was first recognized fifty years ago but up until five years ago only the chronic disseminating form—coccidioidal granuloma—which has a

mortality of 50 to 60 per cent, was recognized.

In 1937 Dickson identified the benign primary form of the infection known as valley fever, which is endemic in the San Joaquin Valley of California, in Arizona and probably in other dry and dusty areas. The incidence of the disease will increase as more soldiers are sent to these regions. Transmission is by inhalation of the vegetative stage of the fungus with dust. It is not, so far as is known, transmitted from person to person or from animal to animal.

There are no pathognomonic symptoms of the beginning of the infection. The symptoms resemble those of influenza or pneumonia or a severe cold. The characteristic erythema nodosum occurs in only about 2 to 5 per cent of the cases. The roentgen and anatomic changes resemble those of tuberculosis, and in cases of suspected tuberculosis in which tubercle bacilli cannot be found in the sputum the possibility of coccidioidal infection should be considered. In most of the cases presented in this article *Coccidioides immitis* could be isolated from the sputum and there were positive skin reactions to coccidioidin. Roentgenograms of the authors' 40 cases are given.

The mortality of primary coccidioidomycosis is low as it does not generally become progressive. When it does assume the acute disseminating form it is probably always fatal and may run its course in from five weeks to seven months. A photograph is given of the most virulent case they have seen, in a Filipino, thirty-six years of age. There were multiple miliary verrucous skin lesions over the face, upper trunk and limbs.—*Audrey G. Morgan.*

Attwood, C. J., and Sargent, W. H. Cystic fibrosis of the pancreas with observations on the roentgen appearance of the associated pulmonary lesions. *Radiology*, Oct., 1942, *39*, 417–425.

Cystic fibrosis of the pancreas in infants and young children is not extremely unusual though its diagnosis is. The authors report 4 cases discovered in 125 consecutive autopsies. In only 1 of these cases was the correct diagnosis suspected from the clinical or roentgen findings.

The etiology of the pancreatic lesions is not definitely known but Andersen believes they are due to obstruction of small or large pancreatic ducts. Though no etiological relationship has been proved between the pancreatic and lung changes it is believed that if the association of this pancreatic disease with definite changes in the lungs were more widely known it would be possible to make the diagnosis more frequently from roentgen examination of the lungs. The best clinical method for the confirmation of the diagnosis is the demonstration of the absence of pancreatic trypsin and lipase in the duodenal juice. The lung changes, as described and illustrated in these cases, consist essentially of purulent bronchitis with bronchiectasis and bronchiectatic abscess formation and surrounding pneumonitis. The changes are most marked around the hilum, decreasing in intensity toward the periphery.

The authors' patients ranged in age from three months to two years which is about the usual age for this disease. Therefore it is in this age group that the pulmonary changes described are most apt to be associated with cystic fibrosis of the pancreas.—*Audrey G. Morgan.*

Swineford, Oscar, Jr., and Harkrader, Charles J., Jr. Intrathoracic lipoma. *Ann. Int. Med.*, July, 1942, *17*, 125–129.

Only 25 cases of wholly intrathoracic lipoma have been reported. The authors report a case in a man of sixty-two admitted to the Hospital of the University of Virginia in severe status asthmaticus with congestive heart failure which had lasted for ten days. For twenty-five years he had had asthma whenever he contracted an acute respiratory infection. Attacks had been relieved in 1937 and 1939 by bronchoscopy. On this admission, bronchoscopy was followed by a severe attack of asthma with shock, and the patient remained semi-comatose with frequent periods of Cheyne-Stokes respiration, cyanosis and pulmonary edema for six days and then died.

Autopsy showed a large lipoma filling the base of the right thorax. The lipoma was not necessarily the primary cause of the asthma. Bronchial stenosis is known to be a cause of asthma and in this case there was stenosis of the middle lobe bronchus, apparently caused by inflammation and not by pressure from the tumor. The middle lobe had apparently not functioned for a long time as shown by the absence of pigment, and this suggests further doubt as to whether the asthma was caused by the lipoma. In a collection of 19 cases reviewed from the literature 5 showed some symptoms of asthma.

Roentgenograms taken during the final illness showed a mass above the diaphragm but they did not show the decreasing density of the shadow from the center toward the periphery which is characteristic of intrathoracic lipoma. This finding was seen to a certain degree in roentgenograms taken in 1937.—*Audrey G. Morgan.*

Kornblum, Daniel. Attempt at roentgenographic visualization of the thoracic duct and cisterna chyli; theoretical considerations and preliminary observations. *Radiology*, Oct., 1942, *39*, 395–399.

A large part of the fats absorbed from the intestine passes through the lacteals and thoracic duct. It seemed to the author, therefore, that it might be possible to get a sufficient concentration of iodine in the thoracic duct and cisterna chyli following the ingestion of iodized oil so that they could be demonstrated roentgenographically. He reviews the anatomy of the duct and cistern and describes studies made with iodipin and lipiodol in an attempt to visualize them. But a shadow conforming to the anatomic position and size of the duct was seen in a roentgenogram of the chest in only 1 out of 12 subjects after the administration of iodized oil. Further observations with lipiodol, however, failed to confirm this structure as a part of the thoracic duct. Further tests of the method are in order.—*Audrey G. Morgan.*

Whitcome, Benjamin B., and Scoville, William B. Postoperative chylothorax. *Arch. Surg.*, Nov., 1942, *45*, 747–753.

A case is described in a woman of thirty-nine in which sympathectomy was performed for hypertension. She was extremely emaciated and in the course of the operation the thoracic duct was torn. No attempt was made to suture it but silver clips were placed on both the proximal and distal ends. The proximal end reopened after seven to ten days and repeated thoracentesis was necessary in order to remove the chyle. On the seventeenth day after operation it was decided to reinject the citrated chyle. The patient died within a few minutes of the beginning of the infusion. She presented asthma, dyspnea, cyanosis, chills and shock. Autopsy was not permitted so it was impossible to say definitely whether death was caused by anaphylaxis, fat embolism, or a foreign protein reaction but the clinical picture indicated anaphylaxis as the cause of death.

This method of treatment has been used in 6 cases of postoperative chylothorax and 3 of the patients have died. They generally die of inanition rather than respiratory embarrassment.

Intravenous injections of fat and protein should be given early in chylothorax to prevent rapid emaciation. If intravenous administration of chyle is used in the future the patient's sensitivity should be carefully tested with an injection of 5 to 10 cc. and the infusion proper delayed for half an hour. The fat globules should be measured microscopically and specimens containing globules over 3 microns in diameter should be rejected; twenty-four hour cultures of the chyle should be made.—*Audrey G. Morgan.*

ABDOMEN

Blackford, Staige D., Bird, Robert M., Jr., and Casscells, S. Ward. Non-operative results in ninety patients with abnormal cholecystograms. *Ann. Int. Med.*, June, 1942, *16*, 1118–1122.

Records of 500 abnormal cholecystograms were found in the files of the Department of Roentgenology of the University of Virginia. Among the patients represented by these, 252 were operated on. Ninety patients not operated on were followed up for an average time of six and a half years. The average age of the patients was 50.3 years. In 25 of the cases the symptoms were mild, in 26 moderate and in 37 severe. In 2 cases the records were not sufficient to grade the severity of the symptoms.

After an average period of six and a half years medical, or rather non-surgical treatment, the results were satisfactory in 47.7 per cent of the entire 90 cases, in 52.5 per cent of those with poor function and 44.9 per cent of those with no function and in 42.8 per cent of those with cholelithiasis.

Conceding the disadvantages of questionnaire follow-up of patients and the small number of the cases, the authors still believe that these results indicate that the patient with uncomplicated cholecystitis should be given a trial on medical treatment and if not promptly relieved he should be operated on.—*Audrey G. Morgan.*

Hoffman, H. S. Benign hepatoma. *Ann. Int. Med.*, July, 1942, *17*, 130–139.

Benign tumors of the liver are very rare and apparently only 58 cases have been described in

the medical literature of the world. They rarely cause symptoms unless they become large enough to cause signs of pressure on the liver or neighboring organs.

The author describes a case in a woman of thirty-eight and reviews 3 cases from the literature. His own patient came for treatment of a mild respiratory infection but said that recently she had been conscious of a sensation in her right upper abdomen, though there was no pain and she had no other symptoms referable to it. She thought she felt a mass in the right upper quadrant. Roentgenograms showed such a mass with a rounded lower pole. They did not show whether it was a prolongation of the liver or a separate mass.

Operation showed a firm nodular tumor projecting from the lower surface of an apparently normal liver. The tumor was shelled out with only moderate loss of blood. Microscopic examination showed liver tissue presenting portal cirrhosis with active hepatitis and hyperplasia of bile ducts.

It is impossible to say whether this tumor developed from a congenital cell rest which later became inflamed or whether an area of focal necrosis became localized as an extrahepatic tumor. None of the cases heretofore described has shown this association of apparently normal liver tissue with an active pathological process in a histoid tumor.—*Audrey G. Morgan.*

BOWERS, RALPH F., LORD, JERE W., JR., and MCSWAIN, BARTON. Cystadenoma of the pancreas. *Arch. Surg.*, 1942, *45*, 111–122.

Descriptions are given of 5 cases of cystadenoma of the pancreas treated at the New York Hospital in the past nine years. The patients were all women, ranging in age from thirty-seven to sixty-six years. Four of them had a history of disease of the bile tract and in 4 of them there was a visible, palpable mass in the left upper quadrant of the abdomen. Roentgen examination showed this mass and a gastrointestinal series showed displacement of the stomach and transverse colon. An intravenous pyelogram in 1 case showed poor function of the left kidney, probably due to pressure of the cyst on the renal artery or vein, as it disappeared after operation. Three of the patients had diabetes which improved after operation in 1 case and was readily controlled by insulin in all.

The splenic vein is dilated and intimately related to the wall of the cyst in these cases. In 1 case injury to this vein during the operation caused the death of the patient. The other patients are all living and well up to as much as two and a half years after the operation.

Photomicrographs show the typical picture of cystadenoma of the pancreas.—*Audrey G. Morgan.*

RABINOVITCH, J., and PINES, B. Cysts of the pancreas. *Arch. Surg.*, 1942, *45*, 727–746.

Seventeen cases of cysts of the pancreas seen at the Jewish Hospital in Brooklyn are described and illustrated with photomicrographs.

Eleven of the cases were pseudocyst, 2 cystadenoma, 2 retention cyst, 1 inclusion cyst and 1 pseudocyst with malignant degeneration, which is rare. Inclusion cyst is a form not heretofore described in the literature. It originates from Brunner's glands included in the pancreas as a result of some defect in embryonic development.

These cysts are usually seen in middle life. The ages of these patients varied from seventy-one to seventy-six years, the average being 45.4 years. Fourteen were females and 3 males. There is some evidence to show that trauma is a factor in the causation of these cysts. Cholecystitis and pancreatitis are also etiological factors.

Diagnosis is rather difficult as the symptoms resemble those of other intra-abdominal tumors. The presence or absence of pancreatic enzymes in the cyst fluid is not conclusive. The patients generally have a history of transient attacks of pain and some of them show loss of weight, constipation or diarrhea, nausea and vomiting and jaundice. The cysts, as a rule, are relatively immobile, though when they are in the tail of the pancreas they may be freely movable. Roentgen examination is of great value. If the tumor is in the head of the pancreas the duodenal curve is enlarged and there are definite changes in the angle of the duodenum. Displacement of the duodenum and small intestine indicates cyst of the pancreas. In 6 of the patients in this series roentgen examination showed a mass in the region of the pancreas.

The correct diagnosis was made in 4 cases before operation; mistaken diagnoses included carcinoma of the pancreas, retroperitoneal tumor, ovarian cyst or fibromyoma of the uterus, peptic ulcer, acute pancreatitis, acute cholecystitis, chronic appendicitis and retroperitoneal sarcoma or cyst of the pancreas.

The ideal treatment is complete removal of the cyst if possible. If the cyst is large and adherent marsupialization is the treatment of choice. The prognosis is good in benign cysts but of course bad in the infrequent cases of malignant cyst.—*Audrey G. Morgan.*

FAUST, D. B., and BRANDSTADT, W. G. A case of pancreatic lithiasis. *Ann. Int. Med.*, July, 1942, *17*, 148–152.

Though 113 or more cases of pancreatic lithiasis have been described only 4 have been diagnosed by roentgen examination before operation, and 30 during operation, as the symptoms are very indefinite.

A case is described in a Negro, aged forty-five, who had been hospitalized repeatedly for "stomach trouble" since 1918. He had severe epigastric pain which at first was not related to the taking of food but later was caused even by the sight or smell of food.

Films of the gastrointestinal series showed a solitary calcified nodule in the liver and fine and medium sized calcified areas in the region of the pancreas. Lateral and oblique views proved the calcifications were in the pancreas.

Surgery was advised but refused, but about eighteen months later the patient had an attack of such agonizing pain that he consented to operation. One small white calculus was removed from the pancreas, but others in the terminal ducts could not be removed. About ten weeks after operation there was a sudden return of pain, probably due to blocking of the pancreatic duct by another stone. Another operation has been advised, though it is doubted whether there is any hope for permanent cure with stones distributed as in this case. —*Audrey G. Morgan.*

PUGH, DAVID G. A roentgenologic aspect of pseudomyxoma peritonaei. *Radiology*, Sept., 1942, *39*, 320–322.

Pseudomyxoma peritonaei is a condition in which masses of gelatinous or myxomatous material, usually originating from a pseudomucinous cystadenoma of the ovary or a mucocele of the appendix, are distributed over the surface of the peritoneum in the form of multiple cysts of various sizes. It has heretofore not been possible to diagnose this condition by roentgen examination but a case is described in a man of thirty-two in whom such a diagnosis was made at the Mayo Clinic. The roentgenograms showed many annular shadows of calcification throughout the abdomen, particularly in the pelvis. Also distributed throughout the abdomen but most marked in the upper part, were shadows of large masses of soft tissue.

Another case is described in a woman of fifty in which the roentgenogram was similar but in her case there was a questionable differential diagnosis from peritoneal paraffinoma. She had had several abdominal operations, and after the last one a bottle of some substance had been poured into the peritoneal cavity to prevent adhesions. It is questionable whether this was mineral oil which sometimes causes a chronic chemical peritonitis. At first the patient seemed to be benefited by roentgen treatment which would indicate pseudomyxoma rather than paraffinoma, but recently she has had no benefit from irradiation.

Roentgen examination is diagnostic in these cases only because of the multiplicity of the cysts and their wide distribution.—*Audrey G. Morgan.*

SIMEONE, F. A. Perinephric abscess. *Arch. Surg.*, Sept., 1942, *45*, 424–442.

The author divides cases of perinephric abscess into simple and complicated. In the former group the infection has reached the perinephrium from distant foci, but cases are also included in which it has reached it by extension from metastatic abscess or carbuncle of the kidney. Complicated abscesses are those that have reached the perinephrium by direct extension from diffuse pyelonephritis or pyonephrosis or by direct or lymphatic extension from adjacent or nearby foci.

Sixty-six cases seen at the Peter Bent Brigham Hospital in the past twenty-eight years are analyzed. If there is fever and pain in the flank perinephric abscess should be considered. Fever, leukocytosis, increased sedimentation rate of erythrocytes, and tenderness in the loin are almost always found. Roentgenograms and laboratory findings are only corroborative. Fixation of the kidney and diaphragm may be seen if the abscess is near the upper pole of the kidney but not necessarily if it is around the lower pole or anteriorly in the perinephrium. Anterior displacement of the kidney has also been reported but an abscess may be anterior and not cause this sign.

In roentgen examination great care must be exercised in exposing lateral films as a very

slight deviation from the true lateral may project the kidney shadow far from its true position and suggest displacement that does not exist.

These patients are generally in poor condition as the primary disease has existed for a long time. At the first operation as little as possible for adequate drainage of the abscess should be done. If drainage of the kidney or nephrectomy is necessary it should be done as a secondary operation.

Among the cases of simple perinephric abscess in this series there was no mortality resulting from the abscess. Two patients died from other causes. For the entire group the mortality was 15 per cent. The mortality in complicated perinephric abscess may be from 14 to 50 per cent. —*Audrey G. Morgan.*

STEINBERG, CHARLES LEROY. The value of splenectomy in Felty's syndrome. *Ann. Int. Med.*, July, 1942, *17*, 26–40.

In 1924 Felty described a disease characterized by polyarthritis of the atrophic variety, fever, secondary anemia, leukopenia, splenomegaly and tachycardia. The disease is a constitutional one with all the symptoms of chronic infection.

Three cases are described and roentgenograms of the joints and photomicrographs of the spleen and bone marrow given. The roentgenograms are very much like those of atrophic arthritis. The bone marrow may be either depressed or hyperplastic. If it is inactive of course splenectomy could be of no value but bone marrow stimulants should be used.

The bone marrow was hyperplastic in 2 of the cases here described. Though cells were produced in abundance by the bone marrow there was apparently some obstacle to their passing into the peripheral circulation. On the theory that this obstacle was the spleen splenectomy was performed in 1 of the cases. Operation was followed by improvement in the blood picture and general health. The arthritis was not affected. Two cases of operation for Felty's syndrome described in the literature were followed by death.—*Audrey G. Morgan.*

GIBB, WILLIAM T., JR. A theory concerning the manner in which the stomach empties itself. *Ann. Int. Med.*, Jan., 1942, *16*, 94–103.

The author reviews the various theories that have been advanced to explain the emptying of the stomach and discusses a theory of his own based on 750 stomach examinations made at St. Luke's Hospital and Knickerbocker Hospital, New York City, with the Wolf-Schindler flexible gastroscope between February, 1938, and September, 1940.

He does not believe that the peristaltic wave is powerful enough to eject the stomach contents. The pylorus is open practically all of the time and closes only for about a second after each emptying of the stomach. Apparently its only function is to prevent reflux from the duodenum.

Gastroscopic examination has shown a new structure in the stomach called the musculus sphincter antri, running at right angles to the long axis of the stomach just opposite the incisura angularis and extending about half way up the anterior and posterior walls. This musculus is not found postmortem and does not seem to be different histologically from the rest of the stomach wall. It seems to be a functional structure with some definite use in the function of emptying the stomach.

When the duodenum is in proper condition to receive the stomach contents or when the stomach contents are in proper condition to be passed on, this musculus sphincter antri contracts forcibly, dividing the stomach into two compartments. At this time what may be termed a systole of the antrum takes place and the liquid food is squirted into the duodenum under considerable pressure.

This theory cannot be proved with the apparatus now available. To prove that during the systole of the antrum hydraulic pressure is increased there as compared with that in the body of the stomach would require something much more sensitive than the double balloon apparatus.—*Audrey G. Morgan.*

BISGARD, J. DEWEY, MATSON, GUY M., and HIRSCHMANN, JEROME. Adynamic ileus and thermal influences on gastric and intestinal motor activity. *J. Am. M. Ass.*, Feb. 7, 1942, *118*, 447–451.

The terms adynamic ileus and paralytic ileus are used to designate a condition characterized by distention and relative inactivity of the bowel. It was found that the Miller-Abbott tube traveled down the small bowel as readily and rapidly in the patient with so-called paralytic ileus as in the patient with normal intestinal function. For a logical explanation in

must be assumed that in a state of ileus the stretching of the bowel wall from distention is in large measure responsible for the lack of motor activity. Another incongruous situation is observed in the relief of ileus which may follow the paralysis induced by spinal anesthesia. By inducing paralysis, spinal anesthesia presumably releases the inhibiting action of the dorsolumbar sympathetic nervous system on the motor activity of the bowel and leaves the vagus unopposed. It is probable, therefore, that ileus results, at least in part, from an imbalance between the antagonistic actions of the dual innervation of the bowel. Complete paralysis of the bowel does occur in the presence of diffuse or generalized peritonitis.

The authors made continuous kymographic recordings of gastric and intestinal motor activity. These recordings registered the variations in pressure exerted on indwelling compressible rubber balloons by the peristaltic contractions of the viscera studied.

In previously reported studies it was shown that ether, nitrous oxide and barbiturates have an inhibiting action, while spinal anesthetics, cyclopropane and avertin with amylene hydrate have either a stimulating effect or no appreciable effect.

A detailed account of the influence of hot and cold applications on gastric and intestinal motor activity and on the secretion of hydrochloric acid was made in a previous communication. In the present study heat applied to the abdominal wall inhibited motor activity of the stomach, the small bowel and the colon, cold stimulated tonus and peristaltic activity. When heat or cold was applied directly to the wall of the stomach by having the patient drink hot or cold water the responses were reversed.

If it is beneficial, as is generally believed, to inhibit the motor activity of the gastrointestinal tract and thereby place it at rest as far as possible in the presence of inflammatory lesions, such as appendicitis and peritonitis, and of bleeding lesions, such as bleeding peptic ulcer, then hot applications and not ice bags are indicated. The increase in gastric acidity which follows chilling of the body surfaces may explain the recurrence of peptic ulcer in the fall and the spring, when there are rapid and radical changes in temperature.—*S. G. Henderson.*

Dudley, Guilford S., Miscall, Laurence, and Morse, Stanley F. Benign tumors of the stomach. *Arch. Surg.*, Nov., 1942, *45*, 702–726.

There are various estimates as to the relative frequency of benign and malignant tumors of the stomach. Among 4,413 autopsies done at Bellevue Hospital in five years 22 per cent of all gastric tumors seen were benign. A comparison of the records of 76,077 adult medical and surgical patients admitted to the same hospital during three years and seven months showed that 456 had been operated on for tumors of the stomach and that only 6 of these, or 1.3 per cent, were benign. This difference between autopsy and clinical records is probably due to the fact that a great many of these tumors cause no symptoms and never come to operation. Probably about 15 to 20 per cent of tumors of the stomach are benign.

The authors discuss 108 cases of benign gastric tumor seen at Bellevue Hospital. Of these, 76 were only found on microscopic examination at autopsy. Forty-two of these patients had been free of symptoms while in 18 the symptoms suggested ulcer, gastritis or malignant tumor. As a matter of fact, these conditions frequently coexist with benign tumors of the stomach. This of course makes diagnosis difficult.

In 30 per cent of the other 32 cases, the diagnosis was made before operation while in the others it was made during operation. Half of the latter group of cases had been diagnosed as carcinoma. The roentgen findings may closely resemble those of carcinoma.

Gastric hemorrhage (seen in 40 per cent of the cases) was the most frequent symptom. It resembles the massive hemorrhage of ulcer rather than the slow bleeding of carcinoma. There was obstruction of the pylorus in about 30 per cent of the cases which was frequently intermittent. In roentgenograms of these cases a filling defect with gastric residue often gave a picture resembling that of carcinoma.

Eight cases of different types of benign tumor of the stomach are described in detail and illustrated with roentgenograms and photomicrographs.

All benign tumors of the stomach should be treated surgically because of the danger of severe hemorrhage and malignant degeneration. Local excision is sufficient for most of the types of tumor but the percentage of malignant degeneration is unusually high in sessile adenomatous polyps at the pylorus. Resection of the stomach is indicated in this group of cases.—*Audrey G. Morgan.*

Poppel, M. H., and Starr, S. Roentgen manifestations of acute infectious mononucleosis in the abdomen. *Radiology*, Oct., 1942, *39*, 437–439.

Acute mononucleosis or acute glandular fever is an infectious disease the causative agent of which is unknown. It occurs mostly in children and young adults and is characterized by fever, enlarged glands and particularly a monocuclear leukocytosis with a reduction of the polynuclear granular cells to from 2,000 to 4,000 per cu. mm. There is a specific agglutination test (herterophile test) which is practically pathognomonic in high titers if the patient has not been given serum.

Abdominal symptoms are not unusual and a case is reported in this article in a male nurse twenty years of age who had been working with another male nurse who had just recovered from mononucleosis. He came to the hospital August 17, 1939, with a history of diarrhea and diffuse pain in the upper abdomen for three days. Roentgen examination showed displacement of the descending duodenum to the right and backward and a pressure defect on the lower surface of the left half of the transverse colon. These findings suggested multiple abdominal masses outside the intestinal tract in the region of the head of the pancreas. Repeated blood counts were normal. On December 3, 1939, a heterophile reaction was first obtained, showing agglutination in a dilution of 1:64. This was thought to prove the diagnosis of acute mononucleosis of the abdominal type. A month later, in January, 1940, a 5 per cent mononuclear count was found.—*Audrey G. Morgan.*

Smedal, Magnus I. Pyloric ulcers. *Radiology*, August, 1942, *39*, 200–207.

Peptic ulcers sometimes occur in the pyloric valve that show certain roentgen characteristics that differentiate them from both duodenal and prepyloric ulcers. This differentiation is very important because these pyloric ulcers are in the great majority of cases benign while the prepyloric ulcers are for the most part malignant. The author does not believe that patients with pyloric ulcer should be subjected to resection of the stomach on the chance of discovering malignant changes which exist in only relatively few of the cases.

To illustrate his point he gives roentgenograms on 10 such cases of acute pyloric ulcer. All showed certain distinctive roentgen characteristics, such as deformity of both the prepyloric area and the base of the cap; distortion and broadening of the pyloric sphincter, especially as healing takes place, and a penetrating type of ulcer crater, usually on the lesser side of the sphincter. All of these cases proved to be benign, which tends to confirm his theory.—*Audrey G. Morgan.*

Poppel, M. H., and Bercow, Celia. Perforation of peptic ulcer. *Radiology*, August, 1942, *39*, 221–225.

Perforation may be the first definite sign of ulceration and is a very serious complication. Perforations may be acute, subacute or chronic. Acute perforation may be shown on a simple abdominal film made in the upright or semi-upright position by the presence of free air under one or both halves of the diaphragm or it may be shown by the presence of free air between the right lateral wall of the abdomen and the liver in anteroposterior films made with the patient in the left lateral recumbent position. But free air under one or both halves of the diaphragm does not necessarily prove perforated ulcer. The differential diagnosis from other conditions that produce the same effect is discussed. The administration of contrast medium is generally necessary for the demonstration of the subacute and chronic forms. Internal fistulae sometimes form gradually from perforation and are discovered by chance on the examination of old people.

A series of roentgenograms is given showing the different types of perforation seen by the authors and illustrating the diagnostic roentgen features.—*Audrey G. Morgan.*

Steigmann, Frederick. Treatment of large gastric ulcers. *Arch. Surg.*, Nov., 1942, *45*, 764–775.

The author is convinced that large ulcers of the stomach should always be operated on. While it is true that only a small percentage of gastric ulcers undergo malignant degeneration, the fact that it occurs at all makes the presence of such ulcers a risk to all patients. Neither clinical, laboratory nor roentgen examination can differentiate accurately between benign and malignant lesions. Achlorhydria may occur in patients with benign gastric ulcer and patients with malignancy may have a free hydrochloric acid up to 40 degrees. Illustrative roentgeno-

grams are given showing the impossibility of differentiation between benign and malignant ulcers by roentgen examination.

But leaving aside the question of malignancy, there is the ever-present danger of hemorrhage in large gastric ulcers. The mortality from such hemorrhages is almost 50 per cent. This is because such bleeding generally occurs in elderly people with sclerotic vessels and the vessels are apt to be embedded in scar tissue which prevents spontaneous contraction. Moreover, these ulcers heal poorly and the patients are rendered chronic invalids. These ulcers may perforate into the abdominal cavity or penetrate into neighboring organs, causing death or anatomic deformities that result in permanent invalidism, and even if they heal they may cause tobacco-pouch stomach or hour-glass stomach which require operation.

Therefore such ulcers should always be operated on at an early stage. This conclusion is based on a study of over 400 cases seen at Cook County and other hospitals in Chicago over a period of ten years.

High gastrectomy is the treatment of choice in the majority of cases. In recent years spinal anesthesia has been used most frequently in these operations.—*Audrey G. Morgan.*

Falk, Victor S. Familial polyposis of the colon. *Arch. Surg.*, July, 1942, *45*, 123–128.

A study is made of a family of 7 children, 6 of whom had congenital polyposis of the colon. The history on the maternal side was negative. The paternal grandfather died at the age of thirty of what was reported as cholera morbus. The father died at forty-eight of carcinoma of the rectum after a long history of diarrhea with blood and mucus which was suggestive of multiple polyposis.

Three of the 6 children affected were males and 3 females. In 2 of them malignant changes developed in the polyps. These patients were both males but they were also the two oldest children. The oldest of the children was thirty and the youngest ten at the time of the examination. The polyps in the ten year old girl were very small.

The 2 oldest patients who showed malignant changes were operated on and died. In the third a successful ileocolostomy was performed but she says she will not be operated on again. In the fourth a permanent ileostomy was established and she will probably submit to total colectomy later. The fifth, a boy of twenty-one, has not yet decided whether he will accept operation and the ten year old girl will be kept under observation until she is older.

The diagnosis in these cases is established by sigmoidoscopy and roentgen examination of the colon. Because of the tendency to malignant degeneration permanent ileostomy should be established and total colectomy and posterior resection of the rectum performed later.—*Audrey G. Morgan.*

Henderson, Samuel G., and Briant, W. W., Jr. The colon in the healthy newborn infant. *Radiology*, Sept., 1942, *39*, 261–272.

The authors studied the colon in 105 healthy newborn infants by means of barium enema, which is very much superior for the purpose to the barium meal. The children were given a cleansing enema in the nursery and then the opaque enema was given by passing a small rubber catheter through a hole in a rubber stopper which is passed 1½ to 2 inches into the anus and held in place by manual pressure while the enema is given. Two to 2½ ounces of barium mixture completely fill the colon of the newborn infant.

Eighty-one of the infants showed definite haustral markings, though usually they were less pronounced than in the adult. Redundancy of the colon was found in almost all cases. A table is given showing the size of lumen, degree of redundancy, position in the abdomen, length and height of the upper flexure in relation to the position of the vertebrae. There was frequently redundancy of the descending colon, splenic flexure, and transverse portion, while the sigmoid was practically always redundant in early infancy and the hepatic flexure almost as frequently. The cecum was found in the right iliac fossa in 50 per cent of the infants; in 25 per cent it was above the crest of the ilium but a true subhepatic cecum was not found. The appendix was seen in 23 cases; the terminal ileum was easily visualized.

Nineteen of the infants were re-examined after three months to one year. Conclusions cannot be drawn from so small a number of cases but redundancy of the colon was less marked in this group, probably because of growth of the body in length. Further group studies should be made of children of different ages in order to follow up the further development of the colon.—*Audrey G. Morgan.*

Bouslog, John S. The normal stomach and small intestines in the infant. *Radiology*, Sept., 1942, *39*, 253–260.

Little attention has been paid to the study of the normal gastrointestinal tract in the infant and young child. In studying the roentgen pattern of the gastrointestinal tract, the author was struck by the differences from the adult pattern. He therefore made photographic studies of the gastrointestinal tract in fetuses and infants who died at birth or shortly thereafter. Photographs, photomicrographs and roentgenograms of such tracts are reproduced.

Anatomists have generally claimed that the stomach of the newborn child lies vertically with the greater curvature to the left and the lesser curvature to the right. It is also claimed that the infant's stomach does not have a fundus. The author's roentgen examinations showed that in the infant the stomach is high in the abdomen, and usually transverse in position with the lower border of the greater curvature above the level of the third lumbar vertebra. The fundus and subdivisions of the stomach can be recognized definitely. The rugae are present and increase in development with the growth of the fetus. The musculature is poorly developed. The duodenum is not yet differentiated. Both the jejunum and ileum are small coiled narrow tubes. The pylorus is well defined. In the infant at birth there is a change in the character of the mucosal folds at the junction of the jejunum and ileum.—*Audrey G. Morgan.*

Deamer, Wm. C., and Capp, Charles S. Clinical aspects of gastro-intestinal disease in childhood. *Radiology*, 1942, *39*, 273–277.

The author discusses the clinical aspects of a number of gastrointestinal conditions in childhood which may be encountered by the roentgenologist, including celiac disease, or chronic intestinal indigestion, Hirschsprung's disease, or hypertrophy and dilatation of the colon, intussusception, peptic ulcer, Meckel's diverticulum, rheumatic abdomen, mesenteric lymphadenitis and abdominal allergy. The discussion is confined to the clinical, not the roentgen findings.

In closing the discussion on the series of articles on gastrointestinal diseases in childhood, Dr. Henderson said he had been asked "What is a normal colon?" and was obliged to answer that he did not know. The question could be answered only by serial studies on the same group of children over a period of several years. The sigmoid tends to change its position in the abdomen from time to time but this can be shown only by serial examinations. As to the question of how long the apparent elongation of the sigmoid persists, none of the children studied were examined after more than a year and he cannot give any statistics on the subject. —*Audrey G. Morgan.*

Spiesman, Manuel G., and Rubenstein, Hyman I. Hodgkin's lymphogranuloma (rectal stricture). *Ann. Int. Med.*, August 1942, *17*, 349–358.

Hodgkin's disease of the gastrointestinal tract is rare and stricture of the rectum caused by this disease is so rare that the case reported here is the third one known. The etiology of the disease is not known.

Hodgkin's rectal structure must be differentiated from carcinoma of the rectum, lymphopathia venereum, ulcerative proctitis and colitis, amebic granuloma, tuberculous and syphilitic stricture of the rectum and post-irradiation stricture.

The authors' case was in a young woman of twenty-four who first came for treatment in November, 1931, for dry cough with no history of respiratory infection. The picture was such as to suggest either leukemia or Hodgkin's disease. Examination of an excised cervical lymph node August 4, 1932, showed the changes typical of early Hodgkin's lymphogranuloma. A biopsy from the perirenal region taken some three weeks later showed a much more advanced stage of the disease.

Deep roentgen treatment was given under which she improved greatly and the next summer (1933) she became pregnant and was delivered of a normal baby on May 17, 1934. Labor and the puerperium were moderately febrile and during the three months following delivery oliguria developed, accompanied by increasing pain in the abdomen and left lumbar region with progresssive edema of the legs. A gastrointestinal roentgen examination did not show anything abnormal.

On August 15, 1943, pyelography showed that the left kidney was not functioning. Glands were found compressing the left kidney and ureter and a biopsy specimen showed the picture of Hodgkin's disease.

Under deep roentgen treatment she improved

and gained weight until the summer of 1939 when she complained of tenesmus and mucosanguineous stools. In March, 1939, rectal examination showed a firm constricting ring in the ampulla of the rectum. Further roentgen treatment was given.

The question arises as to whether the constricting rings were caused by the Hodgkin's disease or the irradiation, but biopsy specimens showed that the Hodgkin's infiltration had extended from the abdominal and pelvic lesions.

From this time the course was retrogressive with fever, abdominal pain, tenesmus and stools containing blood and mucus, followed by progressive emaciation with bronchial congestion. The patient died January 11, 1940, more than eight years after the beginning of the disease. Autopsy was not permitted.—*Audrey G. Morgan.*

Menne, Frank R., Mason, David G., and Johnston, Robert. Lymphosarcoma of the intestine. *Arch. Surg.*, Dec., 1942, *45*, 945–956.

Tumors of the small intestine are relatively rare as compared with those of the stomach and large bowel, due to the anatomic and functional nature of the small intestine.

The authors describe 2 cases of lymphosarcoma of the small intestine. The first was in a man of forty-two who reported that he had had bilious attacks associated with nausea and vomiting since childhood. Later he had constipation necessitating excessive use of cathartics, and for three months before examination he had had a dull ache in the abdomen sometimes increasing to sharp pain. An indefinite mass was felt in the right side of the abdomen. A roentgenogram showed the terminal 8 inches of the ileum somewhat dilated, suggesting partial obstruction. The mucosa of this part of the ileum was mottled, suggesting inflammation. A diagnosis of terminal ileitis was made and operation performed. The mass found in the terminal ileum on operation proved on histological examination to be a very malignant lymphosarcoma. The terminal ileum, the cecum and a part of the ascending colon were removed and the ends of the uninvolved parts of the ileum and colon sutured together. The patient recovered after 155 days of hospitalization and is free of recurrence after four years.

The other patient was a male high school student seventeen years of age, who complained chiefly of flatus and constipation. He had atypical signs of incomplete obstruction, such as tenderness, pain, tympanites, nausea and vomiting, and a diagnosis of intestinal obstruction was made. A tumor of the ileocecal valve removed on operation proved to be a primary lymphosarcoma. The patient died and autopsy showed recurrent metastatic lymphosarcoma of the peritoneum, heart, anterior mediastinum, mesentery and retroperitoneal organs and tissue.

Only about 376 authentic cases of lymphosarcoma of the small intestine have been reported. The authors found only 1 case among 38,076 surgical tissues and 11,446 autopsies. Their second case was seen soon after this investigation was made.—*Audrey G. Morgan.*

Miller, Earl R., and Herrmann, Walter W. Argentaffin tumors of the small bowel; a roentgen sign of malignant change. *Radiology*, August, 1942, *39*, 214–220.

Carcinoids or argentaffinomas are rare tumors that originate in the argentaffin cells, so named because of their affinity for silver, found at the bases of the crypts of Lieberkühn in the gastrointestinal tract. They are generally benign and of no clinical importance. But the authors have seen 3 cases of malignant small bowel carcinoids which they describe in detail, one in a man of fifty, one in a woman of fifty-five and one in a man of sixty-seven.

Generally these patients are middle-aged, though cases have been seen at all ages from ten days up. They complain of long-standing gaseous distention, bloating, periumbilical pain and steadily increasing loss of weight. The roentgenogram shows the small bowel distended with gas and fluid up to a point of partial obstruction. Careful examination at the site of obstruction shows a small filling defect and the bowel is found to be kinked. The diagnosis is suggested by the association of kinking and tumor of the bowel.—*Audrey G. Morgan.*

SKELETAL SYSTEM

Sussman, Marcy L., and Copleman, Benjamin. The roentgenographic appearance of the bones in Cushing's syndrome. *Radiology*, Sept., 1942, *39*, 288–292.

The authors discuss 7 cases of Cushing's syndrome seen at Mt. Sinai Hospital. They were all in adult females, as the majority of cases of this disease are. The clinical features

picture of aleukemic leukemia is associated with extensive destruction of bone.—*Audrey G. Morgan.*

LEVITT, ABEL, and LEVY, DEXTER S. Multiple myeloma. *Ann. Int. Med.*, Nov., 1942, *17*, 863–873.

Multiple myeloma is a form of malignant tumor with multiple foci in the red bone marrow. The etiology is unknown. The majority of cases occur in males between the fourth and sixth decades. The most important method of diagnosis is by roentgen examination of the bones, but the final diagnosis must be made by microscopic study. The blood picture is not characteristic except for a marked anemia.

A case is described in a young man of twenty-five. He was well until September, 1936, when he began to have a dull aching pain in the thighs. He thought this was only the result of over-exertion for about a year, but the pain steadily increased until he was unable to work and became sharp and stabbing. In November, 1938, he was sent to hospital. He presented marked anemia but no Bence-Jones protein in the urine. Roentgen examination showed myelomatous lesions in the skull, lumbar spine, pelvic bones and along the shafts of the femurs. He was kept in bed until his death March 23, 1940, and given treatment for the anemia and other symptomatic treatment. He suffered such intense pain that the use of narcotics was necessary.

Autopsy showed complete disintegration of most of the bones of the body. There was edema of the lower extremities caused by obstruction to venous return by the masses in the pelvis. There were metastatic calcifications of the skin and kidneys, myocardial degeneration and dilatation of the heart and edema of the lungs.—*Audrey G. Morgan.*

MENVILLE, L. J., WILLIAMSON, L., and MATTINGLY, D. Renal rickets, with report of a case. *Radiology*, Oct., 1942, *39*, 410–416.

The first case of late rickets associated with albuminuria was reported by Lucas in 1883. The authors' case reported in this article brings the number of reported cases up to 128. There are two theories as to the cause of renal rickets. One is that the primary causative factor is kidney disease dating from birth or early youth and the other is that it is due to malfunction of the pituitary. The blood calcium is usually decreased in renal rickets and phosphorus increased.

Renal rickets occurs as a rule in late childhood and apparently has no sex preference. There are two types of bone changes, the rachitic type and the woolly type in which, in addition to decalcification, the trabeculae are thin and the metaphyses suggest moth-eaten bone.

The authors' case was in a white boy six years of age who was suffering from disturbed bone formation and severe anemia. For nine months he had had yellow discoloration of the skin and had steadily lost weight and strength and for the past week had been unable to stand. He gradually grew weaker in spite of blood transfusions and infusions of saline and glucose and he died five days after admission. Roentgen examinations had shown decalcification of the bones and calcification of the arteries and soft tissues. The skull showed thickening of the tables, especially at the base. A pyelogram showed no signs of calculi; there was bilateral dilatation of the pelves but the calices were small. Autopsy showed the kidneys small, pale and scarred with several small cysts on the surface and small areas of calcification in the parenchyma. Areas of focal calcification were found throughout the aorta and the common and external iliac arteries. The bones showed erosion of the cortices, decalcification and widening of the haversian canals and fibrosis and giant cells. There were hematopoietic foci in the marrow of the diaphysis of the femur and in the vertebrae.—*Audrey G. Morgan.*

are increased fat on the trunk, vascular hypertension, amenorrhea, purplish stripes on the abdomen, choleric facies, varying degrees of hirsutism, acneiform eruptions on the face and trunk and decreased sugar tolerance. The characteristic roentgen change is osteoporosis. Six of the authors' patients showed osteoporosis, which was most frequent in the skull but most marked in the spine and ribs. In 1 case the femora and humeri were also involved. The osteoporosis of the skull, seen in 5 of the 7 cases, was irregularly distributed, occurring most frequently in the frontal and parietal regions. The osteoporotic areas were roughly triangular in shape, or in some cases roughly circular, resembling cancer metastases.

The osteoporosis of the spine was barely visible in some cases while in others it was so severe as to have caused compression fractures of the bodies of the vertebrae. There was a characteristic change in the anterior ends of the lower ribs near the costochondral junctions. The ribs were expanded to about twice their normal size for a distance of about an inch and this area was outlined by a calcified shell. The roentgen and histological picture was that of callus.

Nearly all cases of Cushing's syndrome reported in the literature are associated with basophilic pituitary adenoma.

Four of the authors' patients presented carcinoma and 3 adenoma of the adrenal cortex, as proved on operation or autopsy.—*Audrey G. Morgan.*

CONNOR, PAUL J., and MAIER, F. JULIAN. The value of roentgen rays in the diagnosis of endocrine diseases. *Radiology*, Sept., 1942, *39*, 283–287.

Endocrine disturbances frequently cause changes in the bones that can be diagnosed by roentgen examination. It is important for the roentgenologist to know the nature of the bone changes in the different endocrine diseases and for all physicians to realize that they must understand the essential facts of endocrinology, no matter what their specialty.

A brief review is given of the bone changes that can be diagnosed roentgenologically in acromegaly, gigantism, dwarfism, both of the endocrine and non-endocrine types, Simmonds' disease, Cushing's syndrome, hyperthyroidism, Addison's disease, hyperadrenalism, hyperparathyroidism, the Schüller-Christian syndrome, disseminated osteitis fibrosa, Paget's disease, mongolism, pineal gland disturbance and hypogonadism. Enlargement of the thymus gland can be demonstrated only by roentgen examination. Ordinarily roentgen examination is of little value in hyperthyroidism but in long-standing cases it may show osteoporosis and often osteomalacia. It may also be possible to demonstrate a substernal thyroid by roentgen examination.—*Audrey G. Morgan.*

BARDEN, R. P., BELK, W. P., PRATT, G. E., and TAYLOR, W. R. [Ewing's endothelial myeloma with extensive skeletal involvement.] *Radiology*, Sept., 1942, *39*, 334–336.

The subject of this clinicopathological conference was a white boy five years of age who had a history of seven months' pain in the joints and intermittent fever. He had been treated for acute rheumatic fever without improvement. Laboratory examinations showed red cells 2,800,000, hemoglobin 50 per cent. The white count was 2,000, of which 68 per cent were adult lymphocytes. Roentgen examination of the skeleton showed many punched out areas of rarefaction in the skull, pelvis and the diaphyses of the femora and tibiae. All the long bones showed smooth, regular periosteal proliferation extending along the shafts. During the next two months enlargement of the spleen, liver and lymph nodes appeared. The child was given supportive and roentgen therapy but with the beginning of the eighth month began to lose ground steadily. Subcutaneous ecchymoses and persistent neutropenia developed. A week before death the white count was 300 with no polymorphonuclears. This agranulocytosis was associated with generalized furunculosis.

A study of the roentgenograms led to a roentgen diagnosis of sympathetic neuroblastoma with extensive bone metastases. A study of the bone marrow, however, showed cells like those of Ewing's endothelial myeloma and the pathological diagnosis was Ewing's endothelial myeloma with extensive bone involvement.

In the discussion Dr. Pratt said that the original blood picture led to a diagnosis of lymphatic leukemia and asked whether a leukemoid blood picture was often seen with bone metastases of tumor. Dr. Belk said he would not say it was frequent but that it might occur in children in extensive replacement of bone marrow with tumor, and it should always be considered in differential diagnosis when the

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THE AMERICAN JOURNAL OF ROENTGENOLOGY AND RADIUM THERAPY

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THE ROENTGENOLOGICAL APPEARANCE OF THE GASTROINTESTINAL TRACT IN SCLERODERMA*

By CLAYTON H. HALE, M.D., *and* RICHARD SCHATZKI, M.D.
BOSTON, MASSACHUSETTS

THE purpose of this paper is to call attention to the fact that scleroderma may produce changes in the gastrointestinal tract, and to describe the roentgenologic appearance of such changes as we have observed them at the Massachusetts General Hospital.

Progressive dysphagia and esophageal disturbances in scleroderma were first reported by Ehrmann[3] in 1903. In 1937, Weissenbach and his collaborators[16,17,18] reviewed the literature and collected 14 cases of scleroderma in which the patients experienced difficulty in swallowing and roentgenologically showed delayed emptying of the esophagus. They also described the roentgenologic appearance of the esophagus and the pathologic changes in 4 cases of their own. More recently Weiss, *et al.*,[15] Jackman,[5] and Ochsner and DeBakey[9] have reported esophageal obstruction in patients with scleroderma.†

While changes in the esophagus associated with scleroderma have attracted the attention of various authors, we have been able to find only one case report as evidence that scleroderma may change the appearance of the small and large intestine. This case was published by Rake[10] of the Johns Hopkins Department of Pathology in 1931. The roentgenologic examination showed dilatation of the small and large intestines and what was thought to be an almost complete obstruction of the descending colon. Dilatation of the colon was confirmed at autopsy but there was no evidence of obstruction. The dilatation was explained on the basis of spasm occurring in the pelvirectal sphincter, although no hypertrophy of the muscles could be demonstrated.

From our own observations it would appear that roentgenologic changes in the intestines, as well as in the esophagus, are not rare, and that they are of clinical importance (Table 1).

AUTHORS' MATERIAL

Study of the Esophagus. During the past five years at the Massachusetts General Hospital we have examined the upper gastrointestinal tract of 22 patients (15 females and 7 males) who were known to have scleroderma, varying in duration from five months to twenty-two years. In 7 of the group, examination of the esophagus was made because of dysphagia. The remaining 15 had no definite symptoms referable to the esophagus; they were examined routinely for the purpose of this study. In 6 of the symptomless group, however, careful questioning disclosed that a sensation

† Published since paper was completed: Lindsay, J. R., Templeton, F. E., and Rothman, S. Lesions of esophagus in generalized progressive scleroderma. *J. Am. M. Ass.*, 1943, *123*, 745–750.

* From the Department of Radiology, Massachusetts General Hospital, Boston, Massachusetts. Read before the New England Roentgen Ray Society, February 19, 1943.

of material remaining in the mid chest, and occasionally localized burning or pain following the hurried ingestion of food, or after eating a large meal, had been noticed. the skin had no correlation with the extent of the changes in the esophagus.

The roentgenologic abnormalities in the positive cases were not absolutely identical

TABLE I

ANALYSIS OF AUTHORS' MATERIAL

Case	Sex	On Admission			Diagnosis by	Esophagus		Intestines		
		Age	Duration of disease	Extent of skin involvement		Clinical symptoms	Roentgen examination	Clinical symptoms	Roentgen examination	
									Small intestine	Large intestine
I	M	20	2 yr.	+++	Clin. Biop.	No	Positive	Yes	Positive	Negative
II	F	28	2 yr.	++	Biop.*	Yes	Positive	Yes	Positive	Not examined
III	M	61	1 yr.	+	Clin.	No	Negative	Yes	Positive	Not examined
IV	F	42	2 yr.	++	Clin.	No	Positive	Yes	Positive	Positive
V	F	22	3 yr.	++	Biop.	Yes	Positive	Yes	Negative	Positive
VI	F	23	2 yr.	+++	Biop.	No	Positive	Yes	Negative	Negative
VII	F	63	6 yr.	+++	Autopsy	Yes	Positive	Yes	Negative	Negative
VIII	F	35	1 yr.	++	Biop. Autopsy	No	Negative	No	Not examined	Not examined
IX	F	14	1 yr.	++	Clin.	Yes	Positive	No	Negative	Negative
X	F	49	4 yr.	++	Clin.	No	Positive	No	Not examined	Not examined
XI	F	64	5 mo.	++	Clin.	No	Negative	No	Not examined	Not examined
XII	M	39	22 yr.	+++	Clin.	No	Positive	No	Negative	Negative
XIII	M	59	1½ yr.	++	Biop.	No	Positive	No	Negative	Negative
XIV	F	56	5 yr.	+	Biop.	Yes	Negative	No	Negative	Not examined
XV	F	46	12 yr.	++	Biop.	Yes	Positive	No	Not examined	Negative
XVI	M	58	12 yr.	++	Biop.	No	Positive	No	Negative	Negative
XVII	F	36	1 yr.	+++	Clin.	No	Positive	No	Negative	Not examined
XVIII	F	11	3 yr.	+	Clin.	No	Negative	No	Negative	Negative
XIX	F	54	2 mo.	+	Clin. Biop.*	Yes	Negative	Yes	Negative	Negative
XX	F	17	5 mo.	++	Clin.	No	Negative	Yes	Negative	Negative
XXI	M	37	2 yr.	++	Clin.	No	Negative	No	Negative	Negative
XXII	M	3	1 yr.	+	Biop.	No	Negative	No	Negative	Negative

* Also dermatomyositis.

Of the 7 patients who complained of dysphagia, 5 showed an esophagus which appeared abnormal roentgenologically; 2 showed no abnormality. Eight patients, who were without subjective symptoms, showed roentgenologic abnormalities. The extent of the sclerodermal involvement of

but they followed a certain pattern. The time of transit of the barium from the pharynx to the stomach was prolonged; in some cases complete emptying of the esophagus was delayed for as much as half an hour. As a rule, the barium passed into the region of the lower esophagus with only

slight delay; when it had reached the lower end of the esophagus it passed into the stomach as a slow but fairly constant trickle. None of the patients showed any difficulty in initiating the act of deglutition, the barium being thrown into the pharynx and upper esophagus in the normal manner. In several patients the lower 2 or 3 centimeters of the esophagus appeared slightly narrower than usual and gave the impression of resisting dilatation beyond a definitely fixed diameter, although the wall itself did not seem to be rigid. Above this area, the esophagus was somewhat dilated. No gross dilatation, however, such as is common in cardiospasm was seen in any of the patients. In 1 case, the esophagus appeared narrower than normal throughout its entire course. In general, there was a tendency for the esophagus to stay open after the main bolus of barium had passed through it, to contain a large amount of air, and for the barium to adhere to the wall longer than would be expected in patients of this age group. The motion of the esophagus appeared to be diminished. In

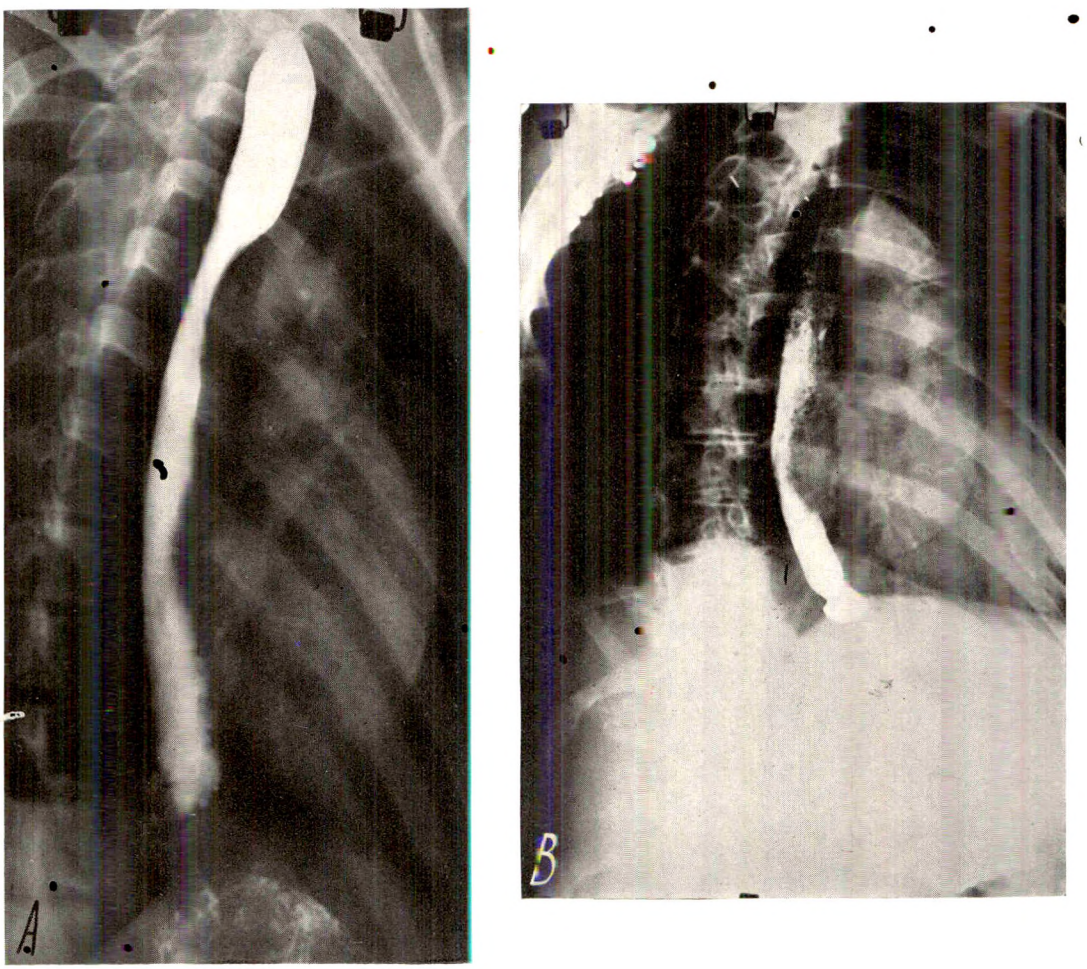


FIG. 1. *A*. Case IV. The upper half of the esophagus is slightly widened. The lower half is somewhat narrow and is still filled with barium at the end of five minutes in the upright position. There is a localized point of constriction 1 cm. above the diaphragm. *B*. Case IX. Roentgenogram taken fifteen minutes after the barium swallow shows the esophagus nearly full of barium. Little if any peristaltic activity was seen.

no patient was there residual fluid or food in the esophagus at the time of the examination.

All the abnormalities noted were more marked in the horizontal position, giving the impression that the esophagus had lost its normal peristaltic force and was relying more or less on gravity for its emptying. In other words, the esophagus had some similarity to the esophagus of an elderly person.

There is a superficial resemblance between the appearance of the lower end of the esophagus in a patient with scleroderma and that of one with cardiospasm, but differentiation is usually possible. In scleroderma we have never seen complete obstruction as in cardiospasm, the barium usually starting to run into the stomach as soon as it reached the cardia. Also in scleroderma, the dilatation of the esophagus was much less marked than that seen in cardiospasm, and no spilling of the barium into the trachea was ever observed. The main difference, however, lies in the fact that the changes in the esophagus do not, as a rule, appear roentgenologically to involve the cardiac end alone, but they involve the entire lower third or half, so far as activity and elasticity of its wall are concerned.

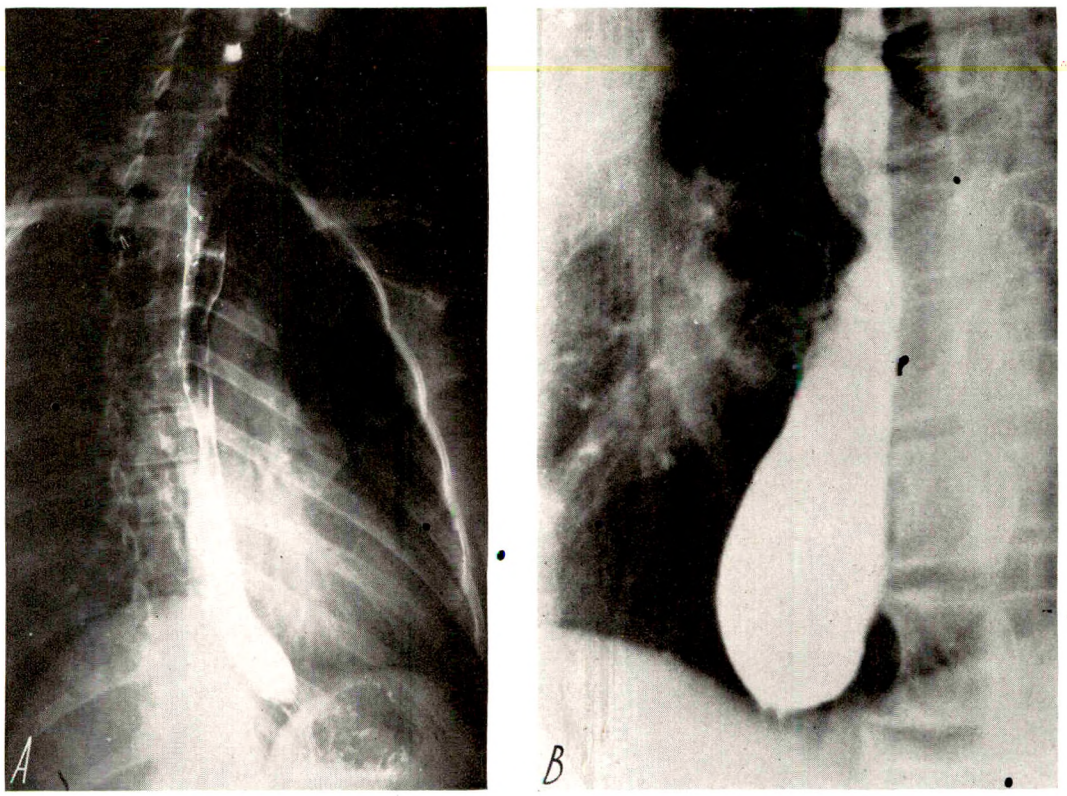


Fig. 2. *A*. Case 11. Barium retained in the esophagus after a period of ten minutes in the horizontal position. The upper half of the esophagus is filled with air and the barium clings to the walls. (This was a finding frequently noted in the positive esophageal cases.) *B*. Case xiii. Roentgenogram taken five minutes after barium swallow in the upright position shows moderate dilatation of the lower third of the esophagus. There was little peristaltic activity.

Study of the Stomach. The stomach was examined in 22 patients. In some cases there was considerable delay in emptying; in some there was spasm of the antrum. The significance of these changes, however, is doubtful.

Study of the Small Intestine. Four of the

18 patients in whom the small intestine was studied showed remarkable changes roentgenologically. In 1 of the 4 cases the intestinal symptoms and the appearance of the intestine stood in the foreground, and the presence of scleroderma was discovered later more or less accidentally, whereas in the 3 other cases scleroderma had been present for many years. Recognition of these changes in the small intestine appears to be of particular clinical importance, and therefore the 4 cases are described in detail.

CASE 1. C. K., a white male, aged twenty, was first seen in August, 1941, with a history of gradually developing pigmentation of the skin over chest, hands and fingers, of two years' duration. For three months, there had been occasional swelling of the fingers and ankles. Physical examination revealed dark pigmentation of the skin covering the entire body, and brawny edema of the face, ankles and several fingers. There was limitation of motions of the hands, fingers and ankles.

This patient's first complaint referable to the gastrointestinal tract, noted at the same time as the beginning of the skin pigmentation, was a so-called sour stomach, characterized by heartburn, gaseous eructations and a sensation of swelling and fullness in the left upper abdomen. Two months before admission he began to have mild, but sharp, cramp-like pains in the epigastrium radiating to the left and downward. The pain frequently occurred at night, especially if he were tired, lasted about thirty minutes, and was at times accompanied by nausea and vomiting. His distress was relieved by applying pressure over the left side of his abdomen, when in the sitting position; it was not relieved by food or alkali. During the two or three weeks just prior to entry the vomiting became more frequent. For several months, the patient had noted that his stools were more fluid than usual; he had three or four partly formed movements a day. There had never been any dysphagia.

Roentgenologic gastrointestinal studies showed slight widening of the lower two-thirds of the esophagus, which remained full of air for a considerable time. There was no definite narrowing at any level. The immediate passage of barium into the stomach was normal but the rate of passage was only a trickle in the horizontal position; the lower third of the esophagus remained filled with barium fifteen minutes after it had been swallowed. In the upright position the emptying was slightly faster, but the barium clung to the esophageal wall for an indefinite period of time.



FIG. 3. Case 1. Roentgenogram taken one hour after barium meal shows the dilatation of the duodenum and upper loops of jejunum.

The stomach was not abnormal. The second and third portions of the duodenum were markedly dilated, measuring 2 inches in width. The barium passed very slowly towards the ligament of Treitz, eventually filling two markedly dilated loops of upper jejunum (Fig. 3). The passage of barium through the small intestine was prolonged, none having reached the ileocecal valve at the end of seven hours. No demonstrable change in the mucosal pattern of the small intestine was noted.

After this examination the question was raised as to whether the atonicity of the upper small intestine could not be explained on the same basis as that seen in the esophagus, namely, as being connected with the patient's scleroderma.

A barium enema showed an essentially normal rectum and colon. Reflux barium from the cecum filled what appeared to be a dilated terminal ileum. Several days later the patient was re-examined following therapeutic doses of

mecholyl but the findings were essentially unchanged. He was discharged with only slight improvement in his epigastric symptoms and no change in the appearance of his stools. He was followed in the Out-Patient Department for the next year, during which time attempts were made to alleviate his gastrointestinal complaints by different dietary regimens, as well as therapeutic doses of nicotinic acid and vitamin B complex in the hope that his diarrhea might be due to vitamin deficiency. At the time he was last seen, November, 1942, his condition was unchanged.

Summary of Case I. A patient with extensive scleroderma and characteristic esophageal changes showed dilatation of the duodenum and upper loops of jejunum and marked delay in transit of barium through the small intestine.

Case II. L. T., a white female, aged twenty-eight, was admitted in August, 1941. She gave a history of having had considerable epigastric distress and gaseous eructations, without relation to meals, for the preceding two years. She often experienced a sensation as of regurgitated stomach contents remaining in the lower esophagus. The symptoms were attributed to aerophagia; antacid treatment gave her almost complete relief.

One year later she noticed for the first time tanning of both sides of her forehead, out of proportion to the amount of sun exposure she had had. Other exposed parts of her body showed similar but less marked pigmentation. Shortly after the appearance of the skin changes, weakness of the shoulder and hip girdles, followed by numbness of the hands and feet, developed. The diagnosis of Raynaud's disease and scleroderma was made, and bilateral sympathectomy was done. Biopsy showed scleroderma and dermatomyositis.

Roentgenologic examination showed that the esophagus did not distend to the size seen in the average patient. There was no obstruction or localized area of narrowing at any point, but the emptying time was prolonged. There was slight fixation of the esophagus as it crossed the left main bronchus and only feeble peristaltic activity was seen throughout. The upper half remained filled with air through which barium could be seen clinging to the mucosa (Fig. 2, *A*).

The stomach and duodenum were normal. The jejunal loops were definitely dilated. Observation was carried out over a period of eight hours, at the end of which time the greater portion of the barium was distributed throughout the jejunum and ileum. There was some gastric residue, and the jejunal loops still showed some dilatation. The mucosal pattern of the visualized small intestine appeared normal.

Two months later the patient was re-examined following therapeutic doses of prostigmine. The appearance of the esophagus, stomach and small bowel, however, was essentially the same as on the first examination. The colon was not examined. The patient was discharged unimproved December, 1941, and has not been seen since.

Summary of Case II. A patient with moderate scleroderma showed prolonged emptying time of the esophagus. The esophagus was slightly narrower than normal and had feeble peristaltic activity. Filling of the dilated loops of jejunum persisted over an observation period of eight hours.

Case III. A. B., a white male, aged sixty-one, entered the hospital in January, 1942, because of nausea and occasional vomiting, slowly increasing in severity, of two months' duration. For two weeks before admission he had vomited at least once each day. He complained of dull, ocasionally cramp-like, fairly constant epigastric pain, starting in the epigastrium and radiating a few inches to the left and downwards. Vomiting gave temporary relief. A more careful search into the history revealed that the patient had felt slightly below par for a year, that his appetite had been poorer than usual, and that his weight had dropped 40 pounds in the year. There had never been any difficulty in swallowing. Thickening and increased pigmentation of the skin over the dorsum of his hands and fingers, which caused moderate limitation of motion, had been present for one year.

Physical examination showed a well developed man in whom signs of weight loss were obvious, although he was not emaciated. Slight epigastric tenderness, but no muscle spasm, was present. He did not appear to be in pain. The hands showed typical scleroderma.

Roentgenologic examination of the esophagus (January 5, 1942) showed no abnormalities. The stomach, which did not appear definitely en-

larged, contained an unusual amount of food and fluid. Barium passed without difficulty into the first and second portions of the duodenum. The third portion was dilated. No barium could be forced into the jejunum during a twenty minute period of roentgenoscopic observation. Later the upper two loops of jejunum were demonstrated. They were dilated, and at the end of four hours no barium was seen to have passed beyond the dilated loops. These findings were interpreted as obstruction of the upper jejunum. No definite point of obstruction could be demonstrated (Fig. 4).

Supportive treatment and Wangensteen drainage failed to alleviate the symptoms, and laparotomy was performed. At operation the proximal 65 cm. of jejunum was found to be of a peculiar grayish color and markedly dilated with individual saccular areas of still greater dilatation, 5 to 7 cm. in diameter, located along the mesenteric side of the jejunum. The wall of the gut was not actually thickened but felt firmer than usual. The peculiar dilated state of the jejunum diminished gradually, becoming normal in size, color and contour approximately 75 cm. from the ligament of Treitz (Fig. 5, *A*). Ten cubic centimeters of 1 per cent novocain was injected into the celiac axis, and immedi-

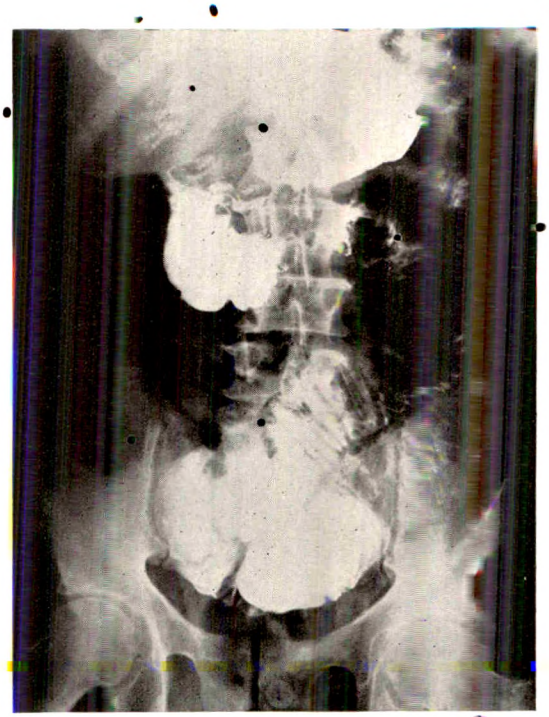


FIG. 4. Case III. Roentgenogram taken four hours p.c. shows dilatation of the upper two or three jejunal loops with no evidence of barium having passed beyond them.

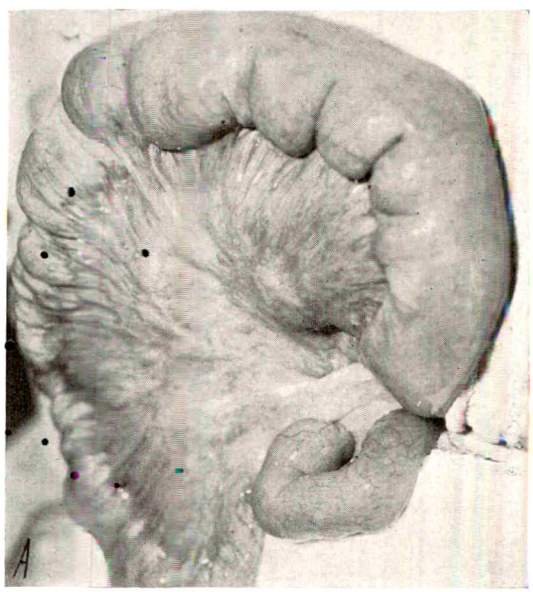




FIG. 5. Case III. *A*, photograph taken during laparotomy showing dilated loop of jejunum. Saccular dilations similar to those shown here are well demonstrated roentgenologically in Figure 7*A*. *B*, roentgenogram taken sixteen days following operation shows an essentially normal appearing gastrointestinal tract. At this time there had been a recurrence of the clinical symptoms.

ately peristaltic and tonic contractions were evident in the dilated loop. Its normal diameter was not restored, however, and the response was transient, disappearing entirely within a few minutes. After some hesitation, the surgeon decided to resect the pathologic appearing area, removing the most proximal 80 cm. of jejunum. *Histopathologic report of resected jejunum:* Edema and hypertrophy of submucosa and interstitial tissue of mesentery, cause undetermined.

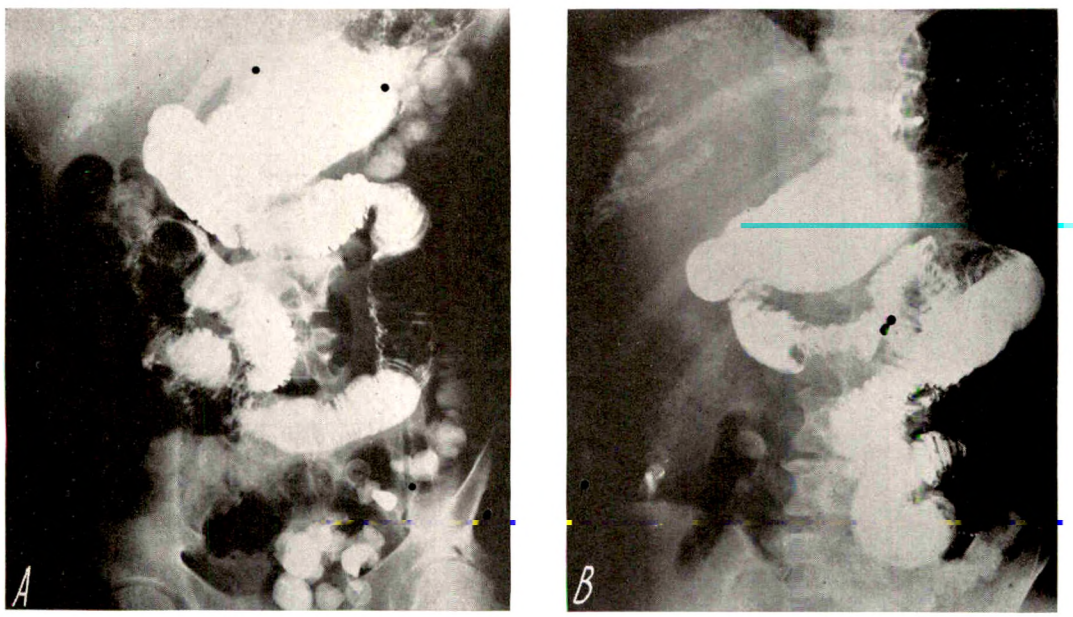


Fig. 6. Case III. *A*, roentgenogram taken on the twenty-fifth postoperative day shows filling of three or four loops of dilated upper jejunum at the end of ninety minutes. *B*, roentgenogram taken after spinal anesthesia shows very little if any change. (The barium seen in the colon was residual from examination made five days before.)

The patient's postoperative course was uneventful for two weeks. On the fifteenth postoperative day, however, he experienced a sensation of fullness in the epigastrium, nausea and heartburn, which he stated were identical with the symptoms he had before operation. The stomach was aspirated, and 650 cc. of black fluid, guaiac positive, were obtained.

Roentgenologic examination of the upper gastrointestinal tract on the following day showed no definite abnormality except slight narrowing at the point of anastomosis (Fig. 5, *B*).

Clinical signs of small bowel obstruction gradually progressed, and five days later, on the twentieth postoperative day, although another roentgenologic examination was still within normal limits, the patient was vomiting several hundred cubic centimeters of fluid each day. He was on constant Wangensteen suction and his entire food intake was parenteral.

On the twenty-fifth postoperative day a fourth roentgenologic examination showed no passage of barium from the stomach to the duodenum throughout thirty minutes of observation. At the end of ninety minutes some barium was scattered throughout the duodenum and upper jejunum. The descending portion of the duodenum was markedly dilated and again definite dilatation of the uppermost loops of jejunum was visible (Fig. 6, *A*). Observations made ten and twenty minutes after spinal anesthesia, which reached the fifth dorsal vertebra, failed to show any appreciable change in the size of the dilated loops, which in the jejunum were 2 inches in diameter.

For the next nine days the patient's course was unchanged. He then developed rather sudden cardiac and respiratory embarrassment, and died within a few hours. A portable roentgenogram of the chest, taken shortly before he died, showed diffuse pulmonary edema.

Autopsy revealed several dilated loops of upper jejunum, which was moderately hyper-

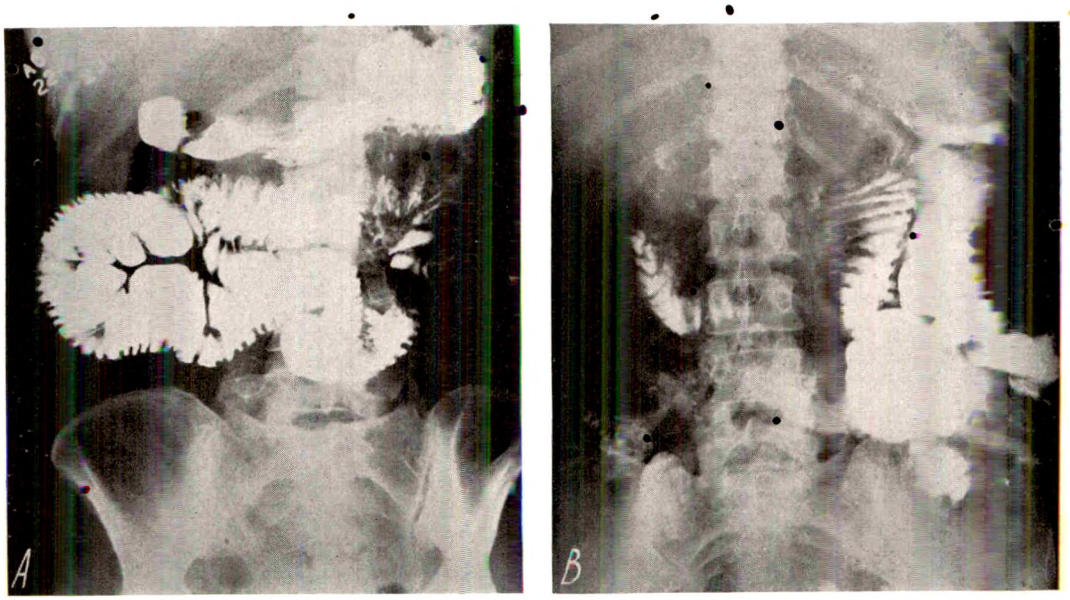


FIG. 7. Case IV. *A*, roentgenogram taken one hour after injection of barium through the Rehfuss tube shows widened loops of jejunum with saccular dilatation. Appearance suggests the sacculation found at operation in Case III, Figure 5*A*. *B*, roentgenogram taken at the end of four hours shows the greater portion of the barium still in the dilated loops of upper jejunum.

trophied. No mechanical cause for the dilatation was seen. The esophagus and colon were not abnormal. The mesenteric lymph nodes were slightly hyperplastic. The serosa and mucosa of the jejunum were not remarkable. The lungs showed extensive pulmonary edema, and in the right lower lobe there was a small, primary oat-cell type of carcinoma, which had been asymptomatic.

Summary of Case III. A patient with minimal scleroderma showed markedly dilated loops of the upper jejunum. At operation this dilatation was confirmed. Resection of the dilated part of the bowel did not prevent recurrent dilatation of the jejunum beyond the area of the original dilatation, and the patient died. Autopsy failed to show any cause for the dilatation of the jejunum.

CASE IV. M. S., a female, aged forty, was first seen in December, 1939, complaining of numbness of the hands and feet after exposure to cold. This had been progressing over a period of two years. She was found to have Raynaud's disease, and bilateral dorsal sympathectomy was done. Early in January, 1940, while convalescing from this operation, she developed right upper quadrant pain and tenderness, with nausea and vomiting. Barium examination of the colon was negative; Graham test was positive, and an acute gallbladder, containing several non-opaque stones, was removed. Postoperative course was uneventful. Considerable temporary improvement in the vasomotor changes of hands and arms followed the sympathectomy.

In June, 1942, she was readmitted because of epigastric discomfort. She had had gaseous eructations and nausea for three months, and diarrhea for two months. Scleroderma had developed in the skin of her hands and arms in the interim.

Roentgen examination of the esophagus showed slight widening of the upper half and absence of peristaltic waves. The lower half appeared slightly narrower than usual with a localized point of more marked constriction about 1 cm. above the diaphragm. A large portion of barium was still in the esophagus after five minutes in the upright position. A roentgenogram taken ten minutes later showed the lower third of the esophagus to be still filled with barium. The upper two-thirds remained open though containing air only. (This patient had not complained of difficulty in swallowing, although she had realized that she could not

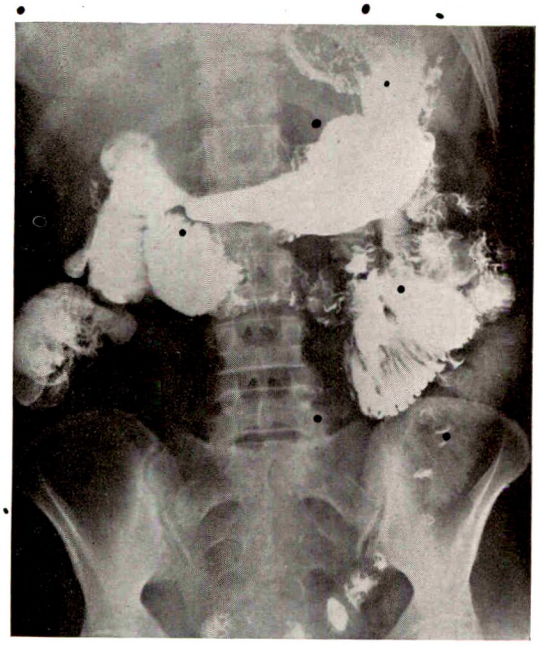


Fig. 8. Case IV. Examination eight months after Figure 7 shows dilatation of the duodenum and upper jejunum and delayed transit of barium. (Roentgenogram taken three hours p.c.) (Barium in the colon, residual from enema examination done the same day.)

swallow as rapidly or as well as other members of her family.)

The stomach appeared normal. There was marked delay in the passage of barium through the small bowel, which showed several widened loops. A few days later a small intestinal enema was done through a Rehfuss tube in the duodenum. The duodenum and jejunum filled slowly, were markedly distended, and showed deep, slow peristalsis. At the end of five minutes only two or three loops of jejunum were filled. After 500 cc. of barium had run into the upper three or four loops of jejunum, over a period of twenty minutes, the Rehfuss tube was removed, and roentgenograms were taken at frequent intervals (Fig. 7, *A*). (For most normal cases, the small intestinal enema leads to filling of the cecum in twenty minutes.[11]) At the end of four hours, the greater portion of the barium was still in the jejunum, whereas parts of the barium were scattered throughout the lower ileum which was also dilated (Fig. 7, *B*). At the end of eighteen hours, about 25 per cent of the barium remained in the distal ileum which was dilated and segmented.

The colon, examined several days later with a barium enema, showed no evidence of obstruction. It did show, however, a peculiar sacculation of the transverse, descending and sigmoid portions, best seen after evacuation (Fig. 10, *B*). The sacculation consisted of localized filling of the haustra, quite common for the appearance of the colon following oral administration of barium but unusual to such a degree following a barium enema. Review of the roentgenograms of the colon taken eighteen months before, just prior to the cholecystectomy, failed to reveal the presence of this saccular appearance.

Proctoscopy disclosed no abnormalities of the rectum or sigmoid.

Re-examination of the upper gastrointestinal tract six weeks later showed no change in the appearance of the esophagus. During the first few minutes only the fundus and uppermost part of the body of the stomach filled; later, the antrum filled but remained spastically narrowed throughout a twenty minute period of roentgenoscopic observation. Barium passed into the duodenum after a delay of about ten minutes, and again the duodenum and upper jejunum appeared definitely dilated. At the end of two hours only the upper two or three loops of jejunum were filled. A roentgenogram taken at six hours showed all the barium still in the jejunum or upper ileum, both of which appeared dilated.

Intensive vitamin treatment was substituted for endocrine therapy, and the patient's diarrhea was relieved for two weeks. At the end of that time, however, the previous bowel habits (five to ten loose, watery movements a day) recurred. Deodorized tincture of opium, the only medication which controlled the diarrhea, kept the patient to three or four partly formed stools daily.

In March, 1943, eight months later, the patient appeared clinically to be in somewhat better condition than she had been when she left the hospital; she was having three to four partly formed stools daily.

Roentgenologic examination at this time showed a normal stomach. The duodenum and upper loops of jejunum were definitely wider than normal and the transit of barium was markedly delayed as before (Fig. 8). Barium enema again showed saccular dilatation in the upper sigmoid, descending and transverse colon similar to that seen in the previous examination. No other abnormality of the colon was

seen; there was a reflux of barium from the cecum into the terminal ileum which appeared slightly dilated.

Summary of Case IV. A patient with moderate scleroderma and definite roentgenologic changes in the esophagus showed marked delay in transit of barium to the cecum, 25 per cent of the meal being still present in the ileum at the end of eighteen hours. Throughout the period of observation dilated loops of small bowel, predominantly in the upper jejunum, were seen. Repeated examinations showed unusual saccular areas of dilatation in the colon.

Discussion. Four of 18 patients with scleroderma examined roentgenologically showed changes in the small intestine. The changes which were noted were quite striking and appeared to be of considerable clinical interest and importance. All 4 patients had clinical symptoms referable to the gastrointestinal tract which indicated partial obstruction. Two other patients had symptoms of gastrointestinal disturbance, but failed to show any roentgen evidence of a pathologic condition.

In the 4 positive cases the roentgenologic appearance was characterized by unusual widening of the part of the small intestine involved, with marked delay in the emptying time of these loops. The dilatation involved the upper small intestine including the duodenum in all 4 patients, and extended downward into the ileum in one. In this particular one the colon also appeared to be abnormal. These roentgen changes must be differentiated from those seen in ileus and in the so-called deficiency diseases. They differed from those of paralytic ileus inasmuch as the pathologic changes usually involved only a certain part of the intestine, and the condition of the patient clinically was never as acute as that found in paralytic ileus. In 1 case, a rather sudden transition from dilated to normal intestine simulated mechanical ileus, but a definite point of obstruction was not found in this or in any of the other 3 cases.

The similarity to severe cases of deficiency disease was striking in some instances. Again, however, the abnormalities seemed to be more localized than is usually seen in that disease. Clinically, the scleroderma patients showed no definite evidence of avitaminosis, and those who were treated extensively with vitamins showed no improvement. Although it is impossible to prove that the changes were not produced by some deficiency, we do not believe that this is the case, and our belief is strengthened by the fact that the changes in the small intestine have some similarity to those of the esophagus seen in the same patients and in other patients with scleroderma.

It is beyond the realm of this paper to discuss the origin of the changes observed in the gastrointestinal tract, but they could be explained on the basis of local changes in the intestines or in the governing nervous mechanism. The negative anatomical examinations in 2 of the patients do not in our opinion definitely exclude the presence of local pathologic changes. On the other hand, the rapid recurrence of changes in the jejunum under our observation in Case III strongly suggests a pathologic condition of the nervous mechanism.

Study of the Large Intestine. Two of the patients with scleroderma showed an unusual appearance in the large intestine which is difficult to explain on the basis of any other pathology, and we therefore assume that it is related to their scleroderma.

CASE V. E. C., an Italian female, aged twenty-two, was first seen in 1930, complaining of alternating redness and blueness of her hands and feet, and of pain in the joints of her extremities. This had been present since the age of nineteen. Skin changes, involving the major part of the extremities, were consistent with scleroderma. The fingers were stiff and showed some flexion deformity. Biopsy of the skin showed scleroderma. Dorsal sympathectomy was performed, but no definite improvement in the vascular changes resulted.

In March, 1937, she first noticed a sensation as of food sticking in the lower portion of her esophagus. Several months later she began to have epigastric distress with occasional nausea

and vomiting, occurring from once or twice a week to once a month, and without relationship to meals or dietary habits. Her symptoms continued unchanged for the next two years.

In February, 1940, *barium examination* of the upper gastrointestinal tract showed definite delay in the emptying of the esophagus with moderate widening of all but the distal 2 cm., which appeared narrower than usual. No peristaltic waves were seen, and at the end of fifteen minutes the lower third was still filled with barium. Above this filled portion the walls were coated with barium and remained open. The stomach was essentially normal.

The patient was discharged, partially relieved, on a controlled dietary regimen. In October, 1942, she returned complaining of lower abdominal cramp-like pain and frequent attacks of diarrhea, four to five liquid stools a day. The difficulty in swallowing and occasional nausea and vomiting were still present.

A barium enema examination in November, 1942, showed the barium to pass more slowly than usual from the rectum to the splenic flexure, and then to proceed at a normal rate to the cecum. The transverse colon appeared to be slightly shortened. There was a peculiar sacculation of the descending and transverse colon (Fig. 9). The areas of sacculation seemed to be the normal colon while the areas in between appeared slightly rigid with irregular outline and change in the mucosal pattern, the mucosa consisting of thickened longitudinal folds only. In many respects the appearance of the pathologic areas was similar to that seen in ulcerative colitis. Re-examination two weeks later showed no change in the appearance of the colon (Fig. 10, *A*).

Sigmoidoscopy failed to show any evidence of ulcerative colitis.

Summary of Case V. A patient with long standing moderate scleroderma and characteristic esophageal changes showed a peculiar appearance of the colon. Areas of sacculation alternated with areas of rigidity similar to those seen in ulcerative colitis.

The second case in which changes in the

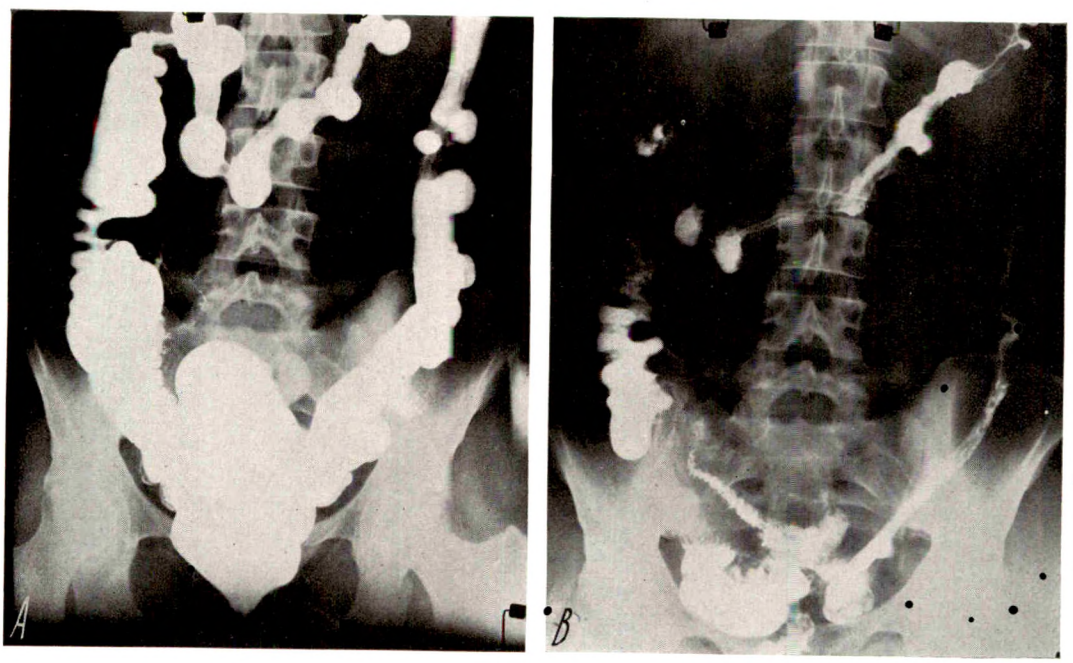


FIG. 9. Case V. *A*, examination of the colon shows slight shortening of the transverse colon with peculiar sacculation of the transverse and descending portions. The areas of sacculation appear to be normal colon while the areas between seem slightly rigid with irregular outline. *B*, post-evacuation roentgenogram shows the mucosa of the narrowed portion of the colon to consist only of thickened longitudinal folds, while the mucosa in the sacculated areas appears normal.

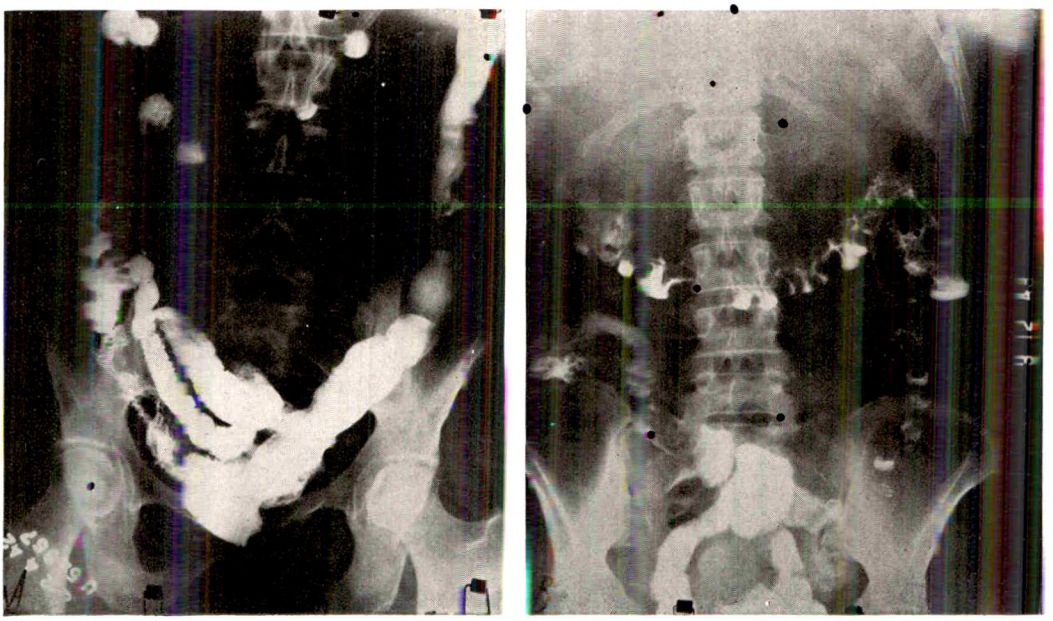


FIG. 10. *A*. Case v. Examination of the colon two weeks after Figure 9 shows essentially no change. Note that the position of the sacculated appearing normal colon is seen in the identical position as before (see Fig. 9). *B*. Case IV. Examination of the colon showed a suggestion of multiple sacculations as seen in Case V. In this case, however, this could be due to localized filling of normal haustra.

colon were found showed changes in the small intestine also (Case IV, described above). In this case the barium enema at a later stage of the disease showed a peculiar tendency towards sacculation but no areas of rigidity were present (Fig. 10, *B*).

The proof of connection between the changes in the large intestine and the scleroderma is by no means convincing. The changes in Case IV were rather minimal or doubtful, and in Case V they could be explained by the presence of ulcerative colitis. In Case V, however, the unusual distribution, together with the absence clinically and sigmoidoscopically of evidence of involvement of the lower part of the colon, justified the mention of these abnormalities and suggested their possible relationship with scleroderma.

SUMMARY

The roentgen appearance of the gastrointestinal tract in 22 patients with scleroderma is described.

Changes from the normal were found to be common in the esophagus (13 cases). They varied somewhat from patient to patient but consisted mainly in delayed emptying, combined with a decrease in the peristalsis of the esophagus.

The small intestine showed significant changes in four patients. There was localized widening, usually of the proximal loops, together with marked delay in the passage of barium through these dilated loops.

There was questionable involvement of the colon in two cases, indicated by a peculiar sacculation, with areas of increased rigidity between the areas of sacculation in one of the cases.

The gastrointestinal changes in scleroderma are of clinical importance. They may simulate cardiospasm, deficiency disease or ileus.

We wish to thank Dr. John H. Talbott for his interest in these studies and for permitting us to use his clinical reports on a number of the cases.

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VOL. 51, NO. 4

THE ROENTGEN DIAGNOSIS OF FETAL MECONIUM PERITONITIS

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THE prompt clinical and roentgenological recognition of intestinal obstruction in the newborn child is of extreme importance. The roentgenographic changes of intestinal obstruction are well known, but we are not aware of any previously reported diagnositic criteria for the recognition of fetal meconium peritonitis that so frequently accompanies stenosis or atresia of the bowel.

According to Abt[1] the first reported series of observations on fetal peritonitis were made by James Y. Simpson who in 1838 described 25 cases. Since then there have been occasional reviews and isolated case reports in the literature. Abt states that although syphilis or bacterial fetal peritonitis may occur, there seems little doubt that meconium peritonitis is the most common form. Markowitz and Loar[4] have defined meconium peritonitis as a non-bacterial chemical inflammation of the peritoneum caused by escape of sterile meconium into the peritoneal cavity through a perforation in the bowel wall. They add that 50 per cent of the bowel perforations occur proximal to a region of bowel atresia. Kornblith and Otani[3] have reported rupture of the bowel in intestinal obstruction due to meconium ileus with resulting meconium peritonitis.

Boikan[2] believes that intestinal peristalsis is necessary for rupture of the bowel and as it is probable that peristalsis does not begin before the fifth month of intrauterine life, so rupture of the bowel probably occurs only after the first half of gestation has been completed. In many instances the site of rupture cannot be found at postmortem examination due to healing over of the perforation.

The clinical manifestations of fetal peritonitis have been reviewed by Abt; it appears that only rarely have the clinical findings led to a correct diagnosis. If intestinal obstruction is associated with the peritonitis, then the symptoms are those of obstruction. It is likely that there have been many cases of survival if the meconium peritonitis was unassociated with bowel obstruction, and the diagnosis suspected only by the dense bands and adhesions disclosed at later operation or autopsy.

Recently we have observed 3 cases in which it was possible to make a roentgen diagnosis of fetal meconium peritonitis by the presence of areas of calcification within the peritoneal cavity. It has long been recognized that calcium deposition occurs within the collections of cornified epithelial cells extruded in the meconium. Linten, cited by Boikan, believes that the calcification may begin as soon as twenty-four hours after the onset of the peritonitis. In each of our 3 cases there was atresia of the bowel and 1 of the 3 cases also had an omphalocele. In each case roentgen examination of the abdomen was made because of suspected intestinal obstruction.

REPORT OF CASES

CASE 1. A three day old white male infant was admitted to the Children's Hospital because of vomiting of two days' duration.

The history shows that the patient was a full term, normally delivered infant that cried well after birth. On the second day of life the patient was given his first feeding and within fifteen minutes vomited. The vomitus contained some bile. The baby continued to spit up small amounts of greenish material. On the day of admission the vomitus became greenish brown in color and vomiting followed every attempted feeding. The abdomen had become somewhat distended during the forty-eight hours preceding admission. The baby allegedly had had

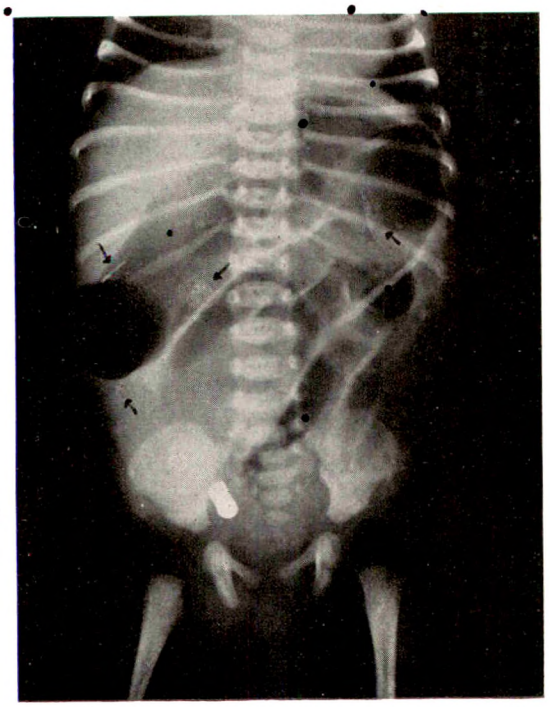


Fig. 1. Case 1. Roentgenogram of the abdomen made in the supine position reveals the dilated loops of small bowel and numerous plaques and streaks of calcific density.

three small stools. There was no cyanosis with the attacks of vomiting.

Physical examination showed a well developed and fairly well nourished, somewhat dehydrated male with a good cry and who moved actively on the examining table. The anterior fontanelle was soft; the neck was not stiff and the heart and lungs appeared clear. The abdomen was slightly distended and tympanitic throughout. There was moderate intestinal patterning over the upper abdomen with visible peristaltic waves. There were no palpable organs or masses within the abdomen. Rectal examination revealed no abnormality. Immediately after the rectal examination the baby passed a small black stool that was waxy in consistency and did not present the usual appearance of normal meconium.

Roentgen examination was made in the lateral supine and upright positions. The roentgenograms showed moderate distention of the stomach and marked distention of the proximal small bowel with numerous air and fluid levels giving the usual appearance of obstruction of the jenunum or proximal ileum. Scattered throughout the peritoneal cavity and lying outside of the dilated loops of bowel were many irregular shadows of calcific density.

The infant was operated upon by Dr. Robert Gross two hours after admission to the hospital. When the abdomen was opened an atresia of the jejunum was disclosed with marked dilatation of the bowel proximal to the point of atresia and the bowel was collapsed distal to it. Scattered throughout the peritoneal cavity were many pinhead-sized yellowish lesions and lightly adherent to the omentum were several larger patches of yellowish material having the appearance of old meconium. There were a few fine adhesions about some of the dilated loops of small bowel. There was no evidence of old perforation, but an extensive search was not made. Several of the collections of yellowish material were removed for study. A side-to-side anastomosis was made around the area of atresia.

The postoperative course was not eventful and the patient is living and well.

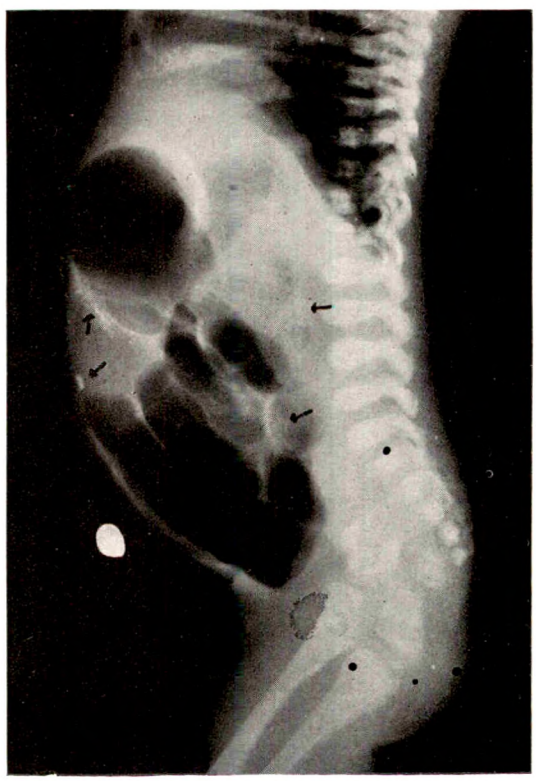


Fig. 2. Case 1. Roentgenogram of the abdomen made in lateral decubitus showing the markedly distended loops of bowel and streaks and patches of calcified meconium.

Pathological examination by Dr. Sidney Farber showed that the sections of the yellowish material removed at operation were made up of stratified, desquamated epithelium, similar to the cells of the same sort found in the contents of the amniotic sac. There were also some fat laden cells, and in and around the epithelial cells collagen was being deposited. Some coagulation necrosis and hyalinization of the cells was taking place. There was an ingrowth of a large number of blood vessels about the masses of cells and some fibrous tissue. Special stains showed that the masses were heavily infiltrated with calcium which appeared to be deposited on all of the stratified epithelial cells. The pathological diagnosis was meconium peritonitis.

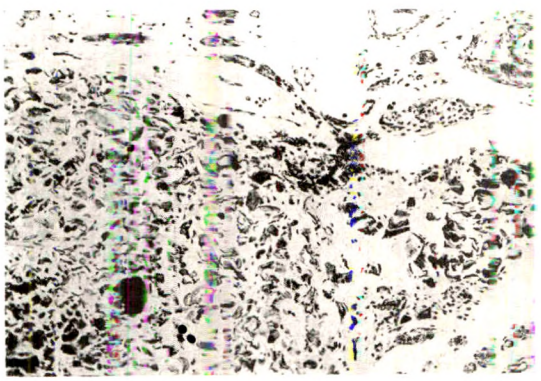


Fig. 3. Case I. Photomicrograph of a portion of meconium plaque. The great majority of the cells are cornified epithelial cells.

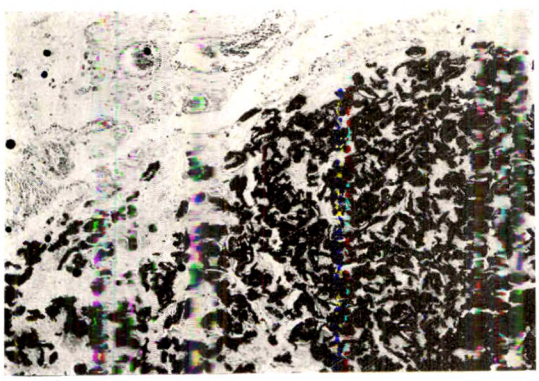


Fig. 4. Case I. Photomicrograph after staining with von Kossa's stain shows the extensive distribution of the calcium encrusting the cornified epithelial cells.

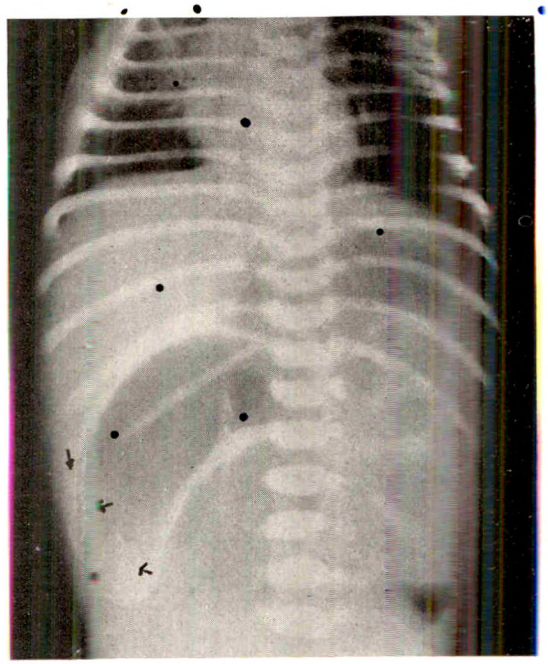


Fig. 5. Case II. Roentgenogram of the abdomen made in the supine position shows dilated loops of small bowel with a single large and irregular collection of calcium in the right lower quadrant.

Case II. A five day old white male infant was admitted to the Children's Hospital because of vomiting since birth.

History revealed that the infant was a full term, normally delivered infant that weighed 7 pounds, 7 ounces at birth. On the first feeding the baby vomited and continued to vomit after every feeding until the fourth day of life when he began to have frequent vomiting unassociated with feedings. At first the vomitus was bile stained, but soon became brownish in color. Abdominal distention was not noted until the day of admission. The baby had had no bowel movements.

Physical examination revealed no abnormalities except slight dehydration and a distended tympanitic abdomen with intestinal patterning.

Roentgen examination was carried out shortly after admission. The roentgenograms disclosed marked dilatation of small bowel loops with numerous air and fluid levels and no evidence of gas in the colon. The appearance was that of a low ileal obstruction. In the right abdomen was a fairly large, but rather faint shadow of calcific density. It could not be made out whether this was within or outside of the intestinal tract.

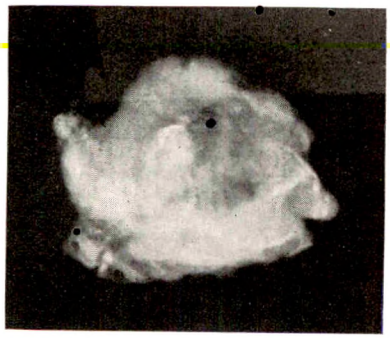


Fig. 6. Case II. Roentgenogram of the specimen removed at operation shows the calcium deposition about the wall of the atretic bowel.

Operation by Dr. Gross revealed an atresia of the ileum about 10 cm. proximal to the ileocecal valve. The peritoneum showed evidence of a diffuse fetal peritonitis with numerous dense and vascular adhesions between the loops of bowel and between the bowel and the parietal peritoneum. Several yellowish plaques of material were imbedded in the adhesions. Attached to the distal loop of ileum was a mass of yellowish material surrounded by adhesions. This was excised. A lateral anastomosis was done around the area of atresia. No evidence of recent perforation could be found.

Pathological examination by Dr. Farber revealed a picture similar to that observed in the first case, but in addition there was a small piece of bowel which also contained stratified epithelial cells with a heavy encrustation of calcium. A pathological diagnosis of fetal meconium peritonitis was made.

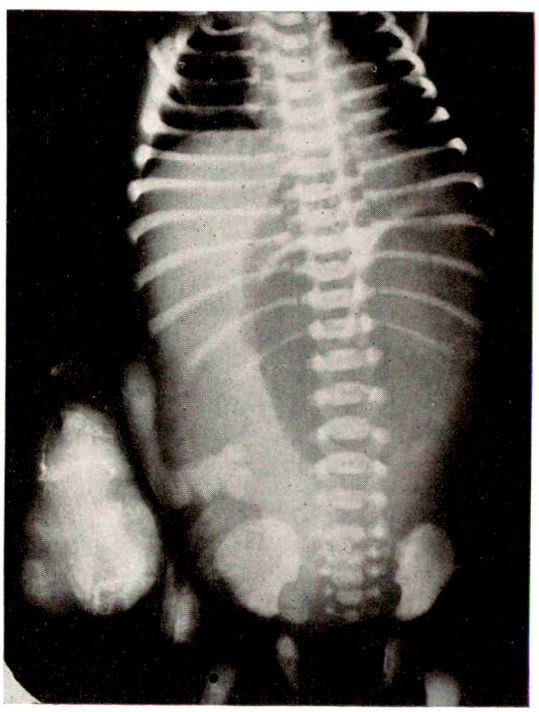


Fig. 7. Case III. Roentgenogram of the abdomen made in the supine position shows a single dilated loop of small bowel and the large omphalocele with the ribbon-like pattern of calcium within.

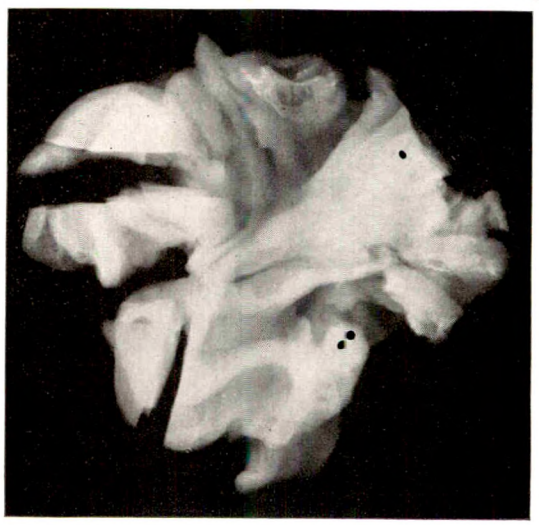


Fig. 8. Case III. Roentgenogram of the excised and opened omphalocele shows the dense adhesions and the calcium deposit outlining portions of the bowel.

The course subsequent to the operation was stormy and after a second operation for the lysis of adhesions the patient died. Postmortem permission was not obtained.

Case III. A female infant was admitted to the hospital four hours after an apparently normal delivery because of a large omphalocele.

Physical examination revealed a fairly well developed white female baby in no apparent distress. There were no evident abnormalities except for a large lobulated omphalocele and a single dilated and tympanitic loop of bowel in the upper abdomen.

Roentgen examination revealed the typical picture of duodenal obstruction. The omphalocele showed irregular areas of calcific density and apparently the calcium lined the bowel wall

as the intestinal patterning within the omphalocele could be made out by the deposits of calcium either within the lumen or along the peritoneal surface.

Operation, performed by Dr. Matson, revealed an atresia of the duodenum. The jejunum and ileum were contained within the omphalocele sac. The omphalocele was excised

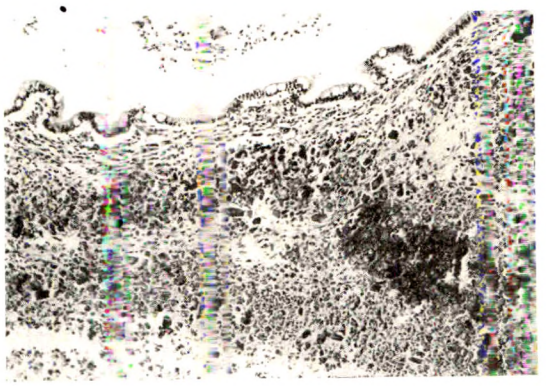


FIG. 9. Case III. Photomicrograph of a portion of the bowel wall shows calcium deposited within the bowel wall.

as the bowel within was strangulated and atretic. The patient survived for only a few hours.

Pathological examination revealed calcium deposition within the lumen of the bowel incarcerated within the omphalocele, within the wall of the bowel, and a small deposit on the peritoneal surface in addition to the extensive fetal peritonitis. There was no evidence of cornified epithelium, as there was atresia of the bowel proximal to the omphalocele.

COMMENT

Three cases of fetal meconium peritonitis have been presented. Each case presented changes on the roentgenogram that we believe are typical of this condition. In each of the 3 cases atresia of the bowel was present giving the usual picture of multiple dilated loops of bowel with air and fluid levels when the roentgenograms were made in the erect position. In addition, there were one or more irregular shadows of calcific density within the peritoneal cavity and either lying within the bowel lumen, within the bowel wall or lying loosely adherent to the peritoneal surface of the bowel or parietes. We know of no other condition that presents a similar roentgen appearance in the newborn child.

SUMMARY

1. Although fetal meconium peritonitis is uncommon we have observed three cases during the past year in which a roentgen diagnosis was possible.

2. Each of the three cases exhibited the characteristic appearance of bowel atresia.

3. In addition there were one or more irregular plaques of calcification visible on the roentgenogram which at operation or autopsy proved to be meconium that had been extruded through a perforation in the bowel and had then undergone calcification.

4. In no case was the site of perforation visible at operation or at autopsy.

5. Perforation of the bowel probably occurred some time before the delivery of the infant, but after the fifth month of intrauterine life.

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INCIDENTAL FINDINGS IN UROGRAMS CONCERNING THE UTERUS

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DURING a study of urograms seen in the Department of Diagnostic Roentgenology of the Cleveland Clinic it was noted that the dye-filled urinary bladders of men and women often differed in shape. The upper surface of the female bladder was usually concave, and that of the male was nearly always convex. This observation led to the conclusion that the concavity of the female bladder is caused by pressure from the uterine fundus. The following quotation from Morris' Anatomy[1] confirms this conclusion: "The anterior surface of the uterus rests upon the upper and posterior surfaces of the bladder. The main support of the uterus comes from the urogenital diaphragm, acting through the bladder, its ligaments serving merely to maintain its horizontal position."

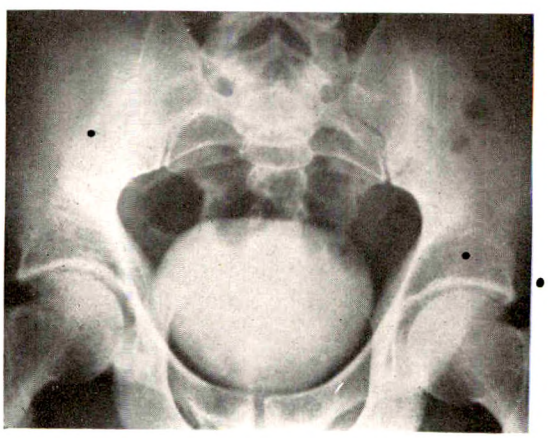


FIG. 1. The normal adult male bladder is round and smooth in contour.

The observation of the concavity in female bladders suggested that useful information about the size and shape of the uterus might be obtained from urograms. The only report in the literature was an article by Snow[2] concerning cystograms as an aid in the diagnosis of extrauterine pregnancy. He stated that the urinary bladder filled with an opaque medium showed a pressure defect on its upper wall "by the uterus which was smaller than it should have been for the period of gestation. From such studies, it is possible to tell whether the uterus is only slightly increased in size or sufficiently enlarged as to contain a full term fetus."

A similar article on "Roentgenologic diagnosis of placenta previa" by Ude, Weum and Urner[3] appeared in 1934. Since then others have confirmed their observations which may be summarized as follows: In placenta previa the placenta is interposed between the upper portion of the maternal urinary bladder and the fetal skull, thus increasing the distance between the bladder and the skull from an average of 6 to 8 mm. to as much as 3 to 5 cm., which may be measured on cystograms.

To confirm the hypothesis that the fun-

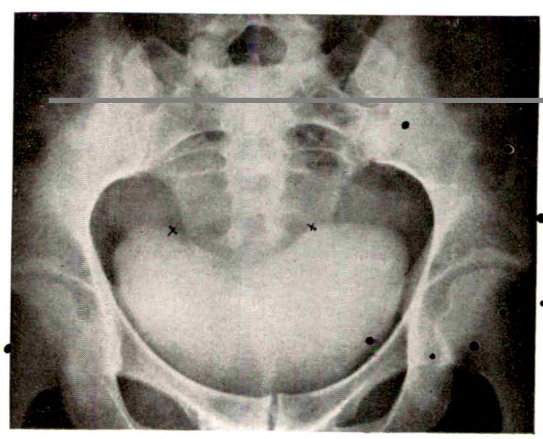


FIG. 2. The normal adult female bladder sometimes has a smooth concavity in the upper surface which is an impression made by the uterus.

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dus of the uterus often makes an impression on the bladder and that its size can be measured, 50 urograms of men were studied to determine the usual shape of their bladders. The majority of the bladders were convex across the fundus, but a few were flattened or slightly irregular. The usual shape of the male bladder is illustrated in Figure 1. Five hundred unselected urograms of women were then examined to study the impression of the uterus on the bladder, and these findings were correlated with case histories and pelvic examinations. The concave impression made by the uterus on the bladder is illustrated in Figure 2.

In order to measure the width of a uterus, marks were made on the urogram on each side of the concave impression on the bladder at points which seemed to represent the lateral borders. The distance between these points was considered to be the width of the uterine fundus within a centimeter or two. Figures 3 and 4*A* show that a concave impression is made on the bladder by the uterus and that its width can be measured. In both of these illustrations a soft shadow can be seen in the concavity in the bladder which no doubt is made by the uterine fundus and very closely corresponds in width to the pressure defect. The uterine fundus occasionally can be seen even in plain roentgenograms as illustrated in Figure 4*B* which is of the same case as Figure 4*A*.

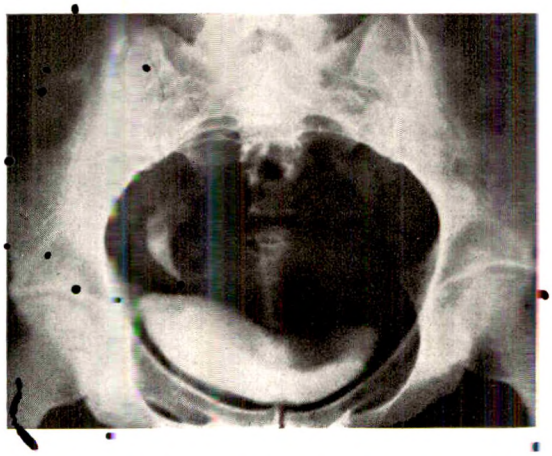


Fig. 3. The shadow of the uterus will sometimes be seen in the concave impression which is not always in the midline of the bladder (see Fig. 4*A* and 5*B*).

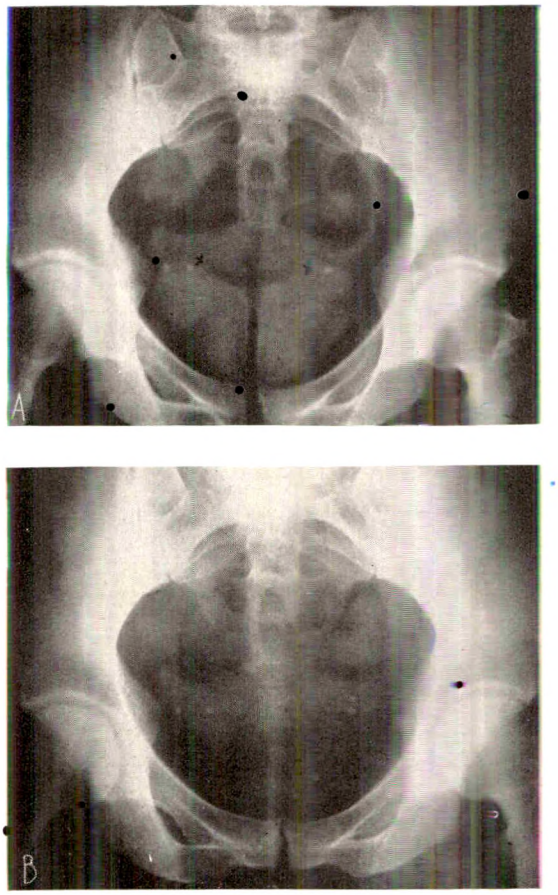


Fig. 4. *A*, the width of the fundus may be estimated by measuring between points at the extreme lateral edges of the uterine impression in the bladder (see Fig. 2 and 7*A*). *B*, the same case as *A*. The shadow of the uterus may sometimes be seen in the plain roentgenogram.

It is also reasonable to assume that the bladder content influences the size of the uterine impression. Figure 5*A* shows a bladder which is very distended, but no uterine impression is visualized. However, it is seen when the bladder is partly emptied as in Figure 5*B*. No doubt this can happen if the bladder is so full that it pushes the uterus backward; then a layer of opaque medium overshadows the uterus. Uterine impressions are not always in the midline of the bladder, as illustrated in Figure 3. When the impressions are not in the mid-

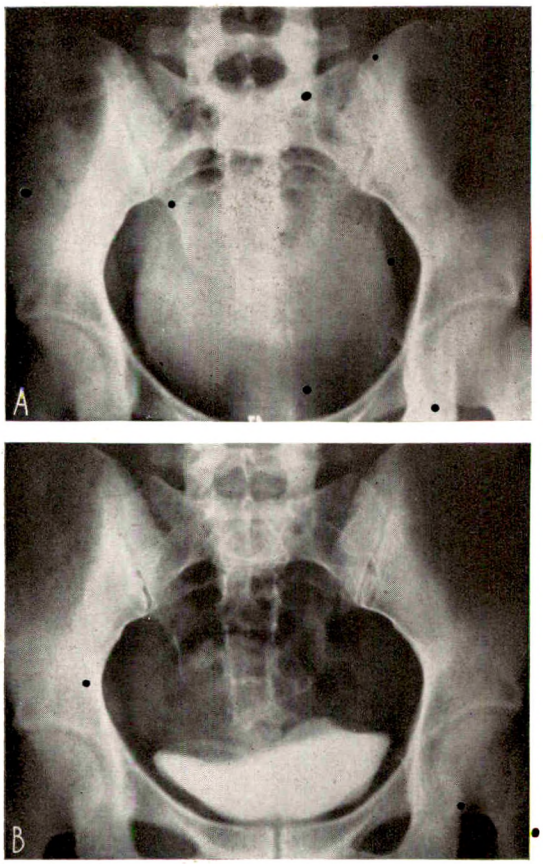


FIG. 5. *A*, no uterine impression may be made if the bladder is distended too much by dye solution. *B*, the same case as *A*. The distended bladder has been partly emptied so that the uterine impression can be seen.

line, the lateral plane of the uterus instead of being perpendicular to the sagittal plane of the body is usually at an angle. This would be expected, because if the uterine fundus moves laterally it would also rotate on its long axis and rest on the sloping surface of the bladder.

The 500 female patients studied were grouped according to the following characteristics:

1. *Menstruating Periodically*. This group was subdivided into (*a*) those showing a uterine impression, (*b*) those showing no impression, (*c*) those with retroversion or retroflexion found by pelvic examination.

2. *Past Natural Menopause*. This group was subdivided into (*a*) those showing an impression, and (*b*) those showing no impression.

3. *Menopause Induced Surgically*. This group was subdivided into (*a*) those having had hysterectomy, (*b*) those having had bilateral oophorectomy.

4. *Not Having Reached Menarche*.

5. *Pelvic Tumors*.

1. Two hundred and ninety-nine of the 500 patients were menstruating periodically and were considered to be in the fertile years of life. Accordingly their uteri should be normal in size, shape, and position.

(*a*) Two hundred and twenty-one of the 299 patients menstruating periodically (74 per cent) showed a uterine impression in the bladder. Seven of them had retroversion. The urogram of special interest was of a woman who had had a uterine suspension. Figure 6 shows the shadow of the uterus, the concavity made by it on the bladder, and also impressions on each side that were probably made by the shortened round ligaments.

(*b*) Seventy-eight of the 221 cases menstruating periodically (26 per cent) showed no uterine impression.

This may be explained as follows: Twenty of the 78 cases had uterine retroversion. The bladders of 4 others were quite full, and 9 had either adrenogenital pathology, very small uteri by palpation,

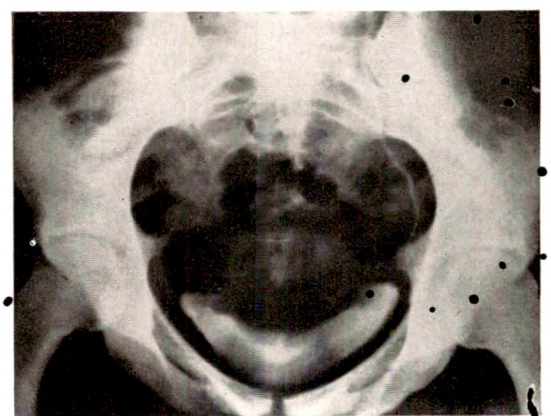
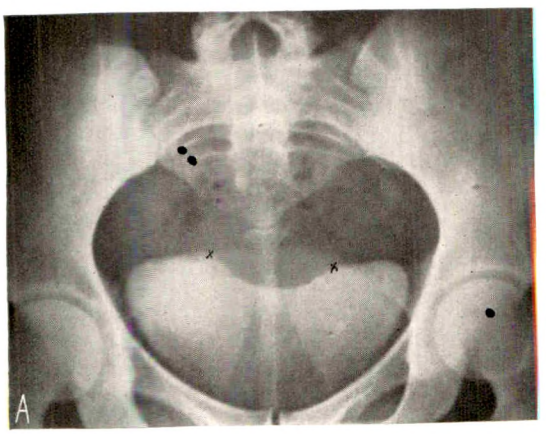


FIG. 6. The shadows of the uterus may be seen in the concave impression in the bladder. Also small impressions on each side laterally made by the round ligaments shortened by uterine suspension.

or were sterile. Therefore, the uteri of 33 of 78 patients (42 per cent) were in retroversion or so small that no impressions were made in the bladder.

(*c*) Twenty-seven of 299 patients menstruating periodically had definite retroversion by pelvic examination, and 20 (74.1 per cent) showed no impression, but the remaining 7 did.

The explanation seems to be that if the uterus is retroverted or retroflexed considerably the fundus does not rest on the bladder, but if the malposition of the uterus is temporary and it returns to its normal location either partially or completely, an impression will be made. If there is only slight retroversion, the uterus should rest more on the posterior surface of the bladder than on the superior surface. Therefore, when the bladder is filled, a thin layer of dye would be visualized anterior to the uterine fundus. This probably does not happen often but may explain the case illustrated by Figure 7*A* which shows a uterine impression and a thin layer of dye anterior to the uterus. Another urogram of the patient shows the uterine impression almost obscured as seen in Figure 7*B*.



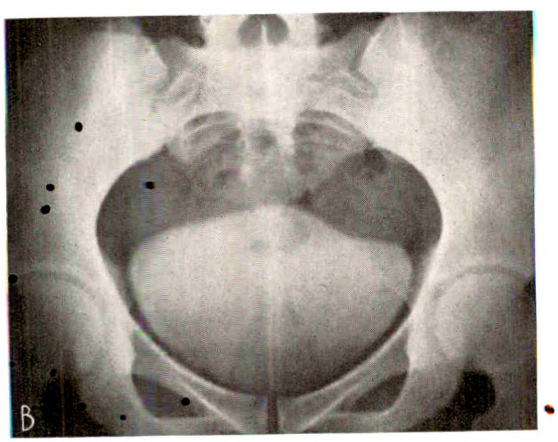


FIG. 7. *A*, if the uterus is in retroversion-retroflexion the impression in the bladder may be small and obscured to some degree by a thin layer of dye solution anterior to the uterus. *B*, the same case as *A* showing a thick layer of dye solution in the bladder almost obscuring the impression of the uterus in retroversion-retroflexion.

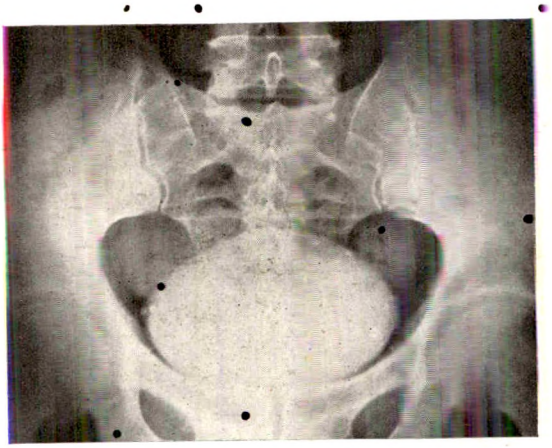


FIG. 8. No uterine impression in the bladder will be made after menopause because the uterus has atrophied.

2. One hundred and twenty-two of the 500 cases had natural menopause.

(*a*) Thirty-three of the 122 cases (27 per cent) past natural menopause showed a uterine impression. The time since menopause of these patients was relatively short and averaged four and a half years.

(*b*) Eighty-nine of the 122 cases (73 per cent) past natural menopause showed no uterine impression, the bladders being flat or convex (Fig. 8). However, the average time since menopause of these patients was ten years, much longer than those showing impressions.

Since 73 per cent past menopause and only 26 per cent menstruating showed no uterine impression on the bladder, the menopause must cause some change in the uterus responsible for this difference. This

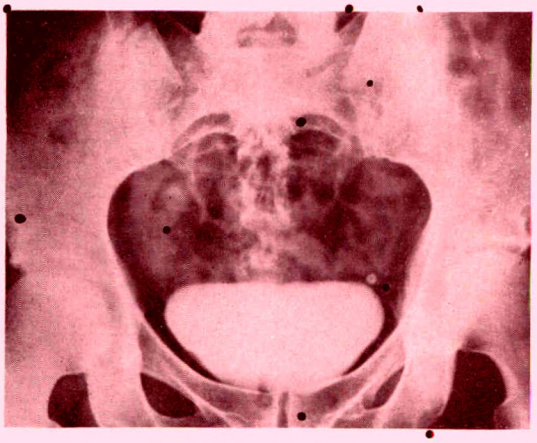


FIG. 9. There is no uterine impression in the bladder after hysterectomy.

may be due either to atrophic changes in the uterus or its ligaments or both. Apparently very little is known about the actual amount and rate of diminution in size of the uterus following the menopause. The observations of Currier[4] help to explain the changes. He referred to women in whom the menopause was progressing, stating that "there is either no perceptible anatomical change, or there is the congestion, the hypertrophy, the granulations of the endometrium, with enlargement of the organ and the occasional profuse hemorrhages which are incidental."

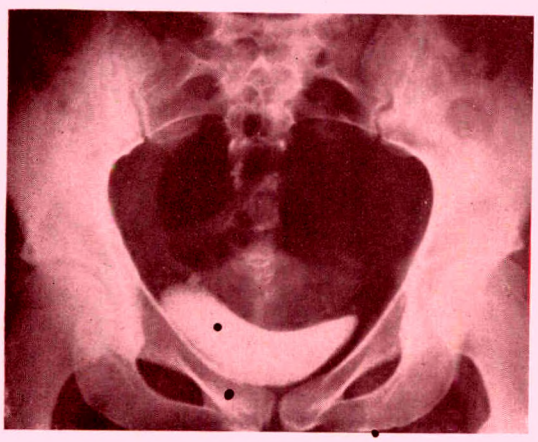


FIG. 10. A uterine impression in the bladder of a woman who had oophorectomy only five months previous to urography. The uterus has not yet atrophied. Its shadow is visualized in the concavity in the bladder.

In those women in whom the menopause has taken place at the customary time as a natural process and in whom two years or more have intervened since the last menstruation, "atrophy of the tissues is well marked—the entire organ is reduced in size."

In those women in whom the menopause has taken place prematurely either from natural causes or from removal of the ovaries, the changes are not materially different from those in whom it took place at the customary time from natural causes,

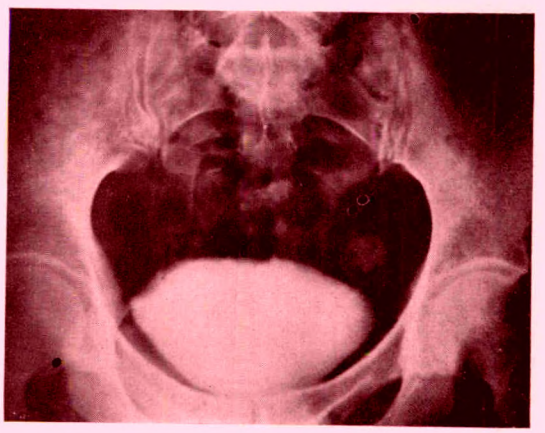


FIG. 11. No uterine impression in the bladder of a woman who had oophorectomy eleven years previous to urography because the uterus has atrophied.

except that the entire process of atrophy moved on more rapidly.

In those in whom the interval of time since the menopause has been very long the corpus may be no larger than a pigeon's egg. On the other hand, even in extreme age the organ has been found to be several inches in length, its nutrition being continued by neoplasm within its structure. In general, in virgins and widows who are continent, uterine atrophy is more complete and more prompt than in married women.

These anatomical changes may explain why no uterine impressions were made in the bladders of such a high percentage of women past the menopause.

3. Forty-nine of the 500 cases had had hysterectomy.

(*a*) All but one of the cases (98 per cent) having hysterectomy failed to show a concavity in the bladder. Figure 9 is of a patient aged forty-three, who had a hysterectomy at the age of thirty-two. One patient said to have had a hysterectomy, had a concavity in the bladder which resembled a uterine impression. It is obvious that if no uterus is present there can be no uterine impression in the bladder, but if a concavity is seen there must be some other cause.

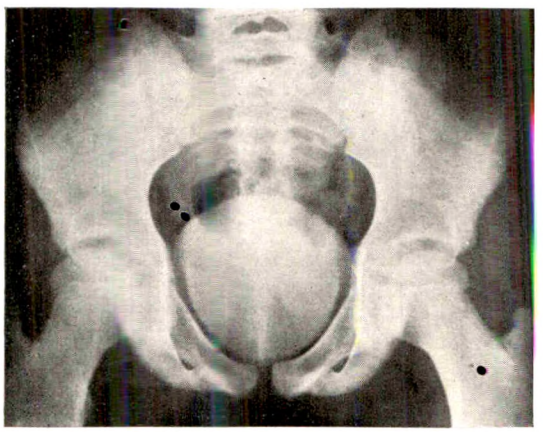


FIG. 12. No uterine impression in the bladder of a girl before menarche because the uterus is not yet large enough.

(*b*) Twelve patients had bilateral oophorectomy, and 10 of these (84 per cent) showed no impression, but 2 did. However, these 2 patients had been operated upon only a few months prior to urography. A few months probably is not time enough for the uterus to atrophy. The shortest interval since oophorectomy of those showing no impression was one year, the average being nineteen years. Figure 10 shows an impression in the urogram of a patient, aged thirty-one, who had had an oophorectomy only five months previously. Figure 11 showing no impression is that of a patient, aged forty-four, who had an oophorectomy and uterine suspension at the age of thirty-eight. No doubt oophorectomy eventually results in uterine atrophy, therefore no impression of the uterus will be made in the bladder.

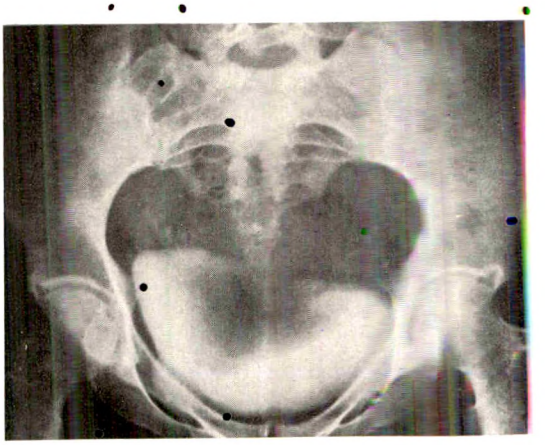


FIG. 13. A deep irregular impression in the bladder made by a large nodular uterine fibroid.

4. Eighteen of the 500 cases had not reached menarche. The average age of this group was ten years. All but one showed no impression and most of the bladders were well rounded, as illustrated by Figure 12. The one patient who showed a slight impression apparently being very near the menarche was ten years old and physically quite mature. Probably her uterus had begun to enlarge, but the impression on the bladder was small. The explanation seems to be that the uteri of these girls were not yet large enough to make an impression in the bladder.

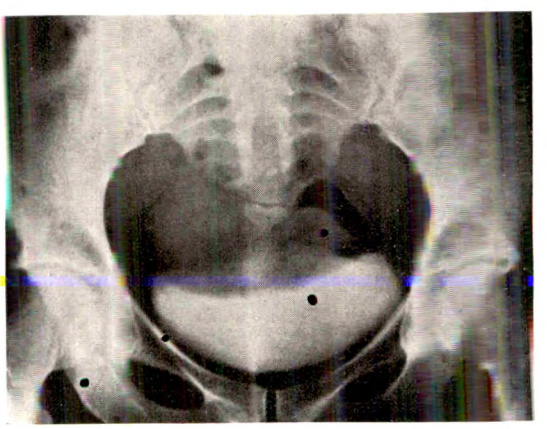


FIG. 14. A wide smooth impression in the bladder made by a diffusely enlarged fibroid uterus.

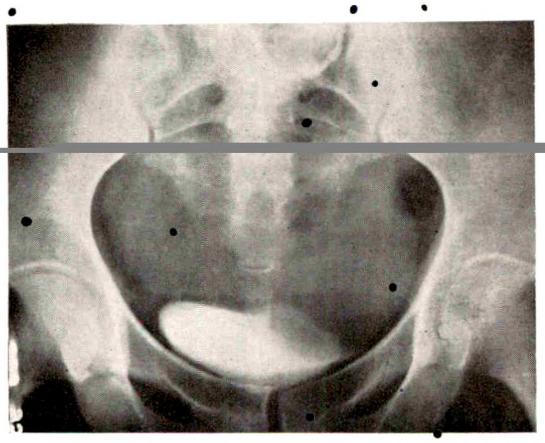


Fig. 15. A deep impression laterally in the bladder made by a nodule of a large uterine fibroid, the shadow of which may be seen filling the pelvis.

5. Cases with pelvic tumors are included in some of the above groups, but a few of special interest will be discussed. The most common tumors were uterine fibroids. Figure 13 shows a medium size fibroid which made a rather deep and irregular impression in the bladder. Figure 14 illustrates a diffusely enlarged uterus making a wide, smooth impression. Figure 15 shows a large fibroid with a nodule compressing the left side of the bladder. The soft tissue shadows of enlarged fibroid uteri also can be seen in Figures 14 and 15. A large ovarian cyst filling the right side of the pelvis and compressing the right side of the bladder is seen in Figure 16.

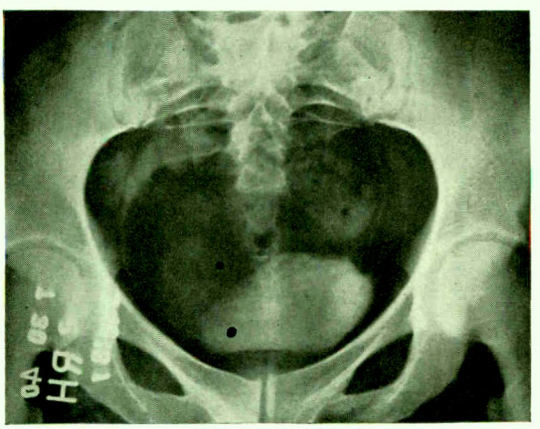


Fig. 16. Lateral compression of the bladder made by a large ovarian cyst, the shadow of which may be seen filling the pelvis.

COMMENTS

This discussion of uterine impressions on the bladder as seen on urograms was based upon cases that primarily were urological problems, therefore few pathological conditions in the pelvis were encountered. It should be possible to give the clinician information about the condition of the uterus or pathological changes in the pelvis as incidental findings when interpreting these roentgenological examinations.

All patients had urograms made at five, fifteen, thirty, and sixty minute intervals. The last gave the most information because the bladder usually contained more opaque medium and the uterine impressions were deeper.

All roentgenograms were made in the supine position, but urograms or cystograms made in the standing position would probably give the most accurate information because the uterus would fall more heavily upon the bladder and make a better impression.

Some patients had manual pelvic examinations made by physicians with experience but not always was there agreement about the size of the uterus. Also it was so difficult to do pelvic examinations on a few patients that even a rough estimate of the size or position of the uterus could not be obtained, but the roentgenological methods described seemed to give accurate information.

In certain cases cystograms may be indicated for the specific purpose of studying uterine impressions, and roentgenoscopy while the bladder is filling might be of value.

CONCLUSIONS

1. From a study of 500 routine urograms of women, it was found that the uterus may make a concave impression on the urinary bladder.

2. The cases were grouped according to the contour of the uterine impressions on the bladder seen on urograms and cor-

related with case histories and the findings from manual pelvic examinations. Percentages are given of the proportion of cases in each group.

3. It was found that the size, shape, and position of the uterus and the presence of pathological conditions in the pelvis could often be determined by the contour of the impression on the bladder seen in urograms.

4. The absence of a uterine impression on the bladder was explained on the basis of (*a*) the uterus being too small, (*b*) retroflexion or retroversion of the uterus, (*c*) absence of the uterus from hysterectomy, (*d*) atrophy of the uterus after natural or induced menopause, and (*e*) the bladder being over-distended with opaque medium.

5. Useful information of clinical significance can often be obtained about the condition of the uterus or the presence of pathological conditions in the pelvis from routine urograms or similar roentgenological examinations.

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THE ROENTGENOGRAM AND SOME CHRONIC NON-TUBERCULOUS PULMONARY CONDITIONS*

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THE frequent insidiousness of the onset of pulmonary tuberculosis, and the complexity and lack of uniformity of the course of the disease, may make the recognition of tuberculosis difficult. The roentgen ray is of great diagnostic import, and, in many cases, may be the only determining factor in the diagnosis of pulmonary tuberculosis. There is no intention on my part to detract from the value of this diagnostic feature which has contributed so much to the early recognition, understanding and management of pulmonary tuberculosis. However, there are cases when roentgen changes in the lungs are observed which appear to be typically characteristic of tuberculosis; subsequently, other important diagnostic adjuncts reveal the absence of tuberculosis and the presence of non-tuberculous lung disease.

CASE REPORTS

The following 5 representative case reports form the basis for this study.

CASE 1. A male, aged twenty-nine, laundry driver, from Chicago, Illinois, was sent to the Sanatorium of the Jewish Consumptives' Relief Society, at Spivak, Colorado, on November 3, 1940, with the diagnosis of active pulmonary tuberculosis of the miliary type.

This conclusion was made on roentgenologic interpretation.

The onset of his illness was with lassitude and malaise in August, 1940, which became progressively worse. Coincidentally, the patient became increasingly pale. On October 7, 1940, he experienced a severe pleuritic pain over the left chest. A roentgenogram revealed involvement of both lung fields and immediate admission to a tuberculosis sanatorium was advised.

Physical examination revealed a well developed and fairly well nourished, but very pale patient. There were no symptoms of active pulmonary tuberculosis or of any respiratory infection. Sputum was negative. A generalized adenopathy, particularly of the cervical, axillary, inguinal and left cubital lymph nodes was present. The spleen was markedly enlarged and the liver only slightly increased in size.

The roentgen picture was unusual (Fig. 1).

The blood examination upon admission revealed the hemoglobin to be 45 per cent. The red blood count was 3,100,000 giving a color index of 0.72 and corresponding to a hypochromic anemia. The total white count upon admission of 5,300 dropped to 4,100. The differential count showed a range of 71–81 per cent lymphocytes and 27–11 per cent neutrophiles. The lymphocytes were small and large, with the small ones definitely prevailing. There were no abnormal polymorphonuclear leukocytes. A sternal puncture revealed only small lympho-

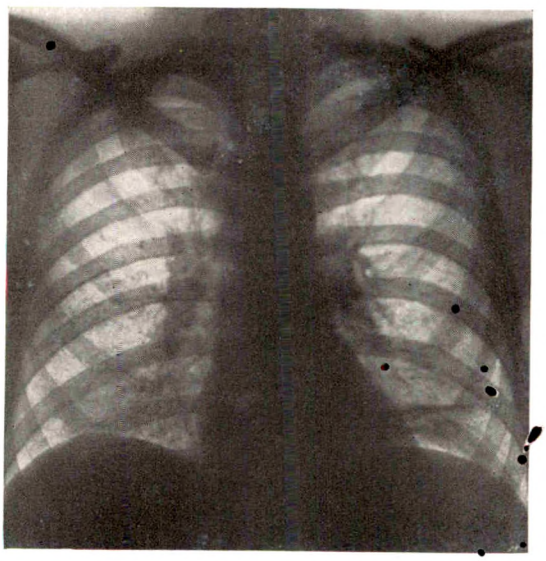


FIG. 1. Case 1. Both lung fields from the first rib anteriorly to the diaphragm are infiltrated with minute areas of densities—the peculiarity is that the apices and the periphery of both lung fields are practically uninvolved, while the infiltration is very heavy above both diaphragms, symmetrically, giving the impression of a butterfly distribution.

* From the Medical Department of the Sanatorium of the Jewish Consumptives' Relief Society, Spivak, Colorado.

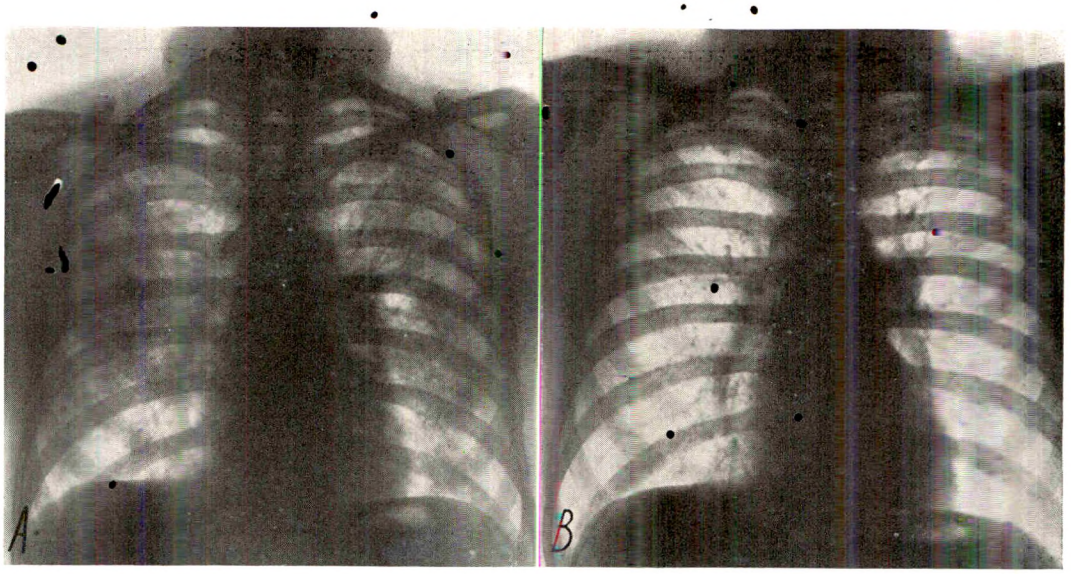


FIG. 2. CASE II. *A*, both lung fields from apex to base are heavily infiltrated with very fine areas of densities—it is almost impossible to distinguish any uninvolved lung tissue. *B*, there has been complete absorption of the massive infiltration with *restitutio ad integrum* and absence of residual deposits.

cytes. The pathologist's report of the biopsy of a cervical lymph node was aleukemic lymphadenosis. In aleukemic lymphadenosis, which was our diagnosis in this case, rather than pulmonary tuberculosis, although lymphosarcoma without lymphemia must be considered, there is a lymphatic infiltration of the peribronchial and interlobar connective tissues, producing the changes noted in the roentgenogram.

CASE II. A male, aged twenty-six, was admitted to our institution on March 10, 1939, from New York City, with the diagnosis of active tuberculosis of the miliary type, made by roentgen examination. Upon admission he was in excellent general condition. Sputum was negative. The roentgenogram gave the impression of a massive hematogenous pulmonary tuberculous spread in both lung fields (Fig. 2*A*). Another roentgenogram taken less than three months after admission showed a complete disappearance of the massive bilateral infiltration (Fig. 2*B*). Because of the rapid disappearance of the massive pulmonary lesion in so short a period of time, a further investigation of the patient's case history was made. It was learned that for eleven years he had worked constantly as a floor man in an embroidery concern, which brought him into persistent contact with, and prolonged inhalation of, a fine chalk dust used as a "stamping powder." An analysis of this powder revealed calcium carbonate. The roentgen changes were due to calcicosis and not to tuberculosis.

CASE III. M. S., male, aged sixty, was admitted to this institution on June 12, 1942, with the diagnosis of active tuberculosis of the right upper lobe. Onset of his illness was in 1935 when he developed a non-productive cough. At that time the diagnosis of minimal right apical tuberculosis was made. His sputum was negative. The patient was on a modified rest regime. In September, 1941, he began to complain of weakness, tiredness and dyspnea. The cough was non-productive. Sputum remained negative.

Upon admission to this institution, he was in fair general condition. His cough and expectoration were minimal. Sputum was negative. He appeared to be very nervous, and complained of marked fatigue. Basal metabolic rate was plus 42 per cent. The roentgen appearance is shown in Figure 3. Because of the persistently negative sputum examinations, a tumor rather than tuberculosis was considered, in addition to the definite diagnosis of hyperthyroidism. The patient entered the Mayo Clinic on August 19, 1942, where a thyroidectomy was performed. The shadow in the right upper lobe subsequently proved to be a Grade 4 adenocarcinoma.

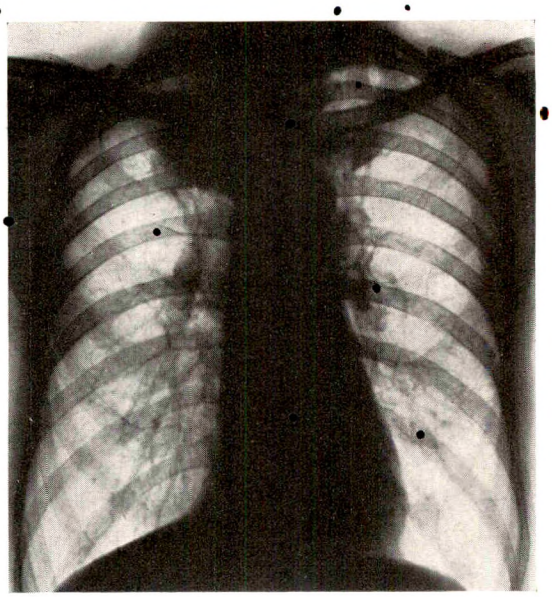


Fig. 3. Case III. There is a dense oval shaped shadow in the right upper lobe extending from the fourth rib posteriorly to the sixth rib posteriorly of the spine. At the inferior border of this dense shadow is a well demarcated nodular area of lesser density attached to the dense shadow and peripherally located.

Case IV. M. E., female, aged fifty-nine, was admitted to this institution on February 28, 1942, with the diagnosis of far advanced bilateral pulmonary tuberculosis. Her history revealed that she had influenza in 1922. A cough which was present at that time had persisted. Two months prior to her admission her cough increased and the expectoration was blood tinged. Numerous consecutive sputum analyses were negative for tubercle bacilli. Upon admission, she was in fair general condition. Her cough and expectoration were slight, sputum was negative. Urine showed 3 plus sugar and blood sugar was 250 mg. Blood pressure was 180/104. Roentgen examination was confusing (Fig. 4). Because of the persistently negative sputum examinations, the diagnosis of pulmonary tuberculosis could not be made with certainty. Diabetes mellitus and hypertension were the absolute findings. Her course in the institution was retrogressive and characterized by numerous hemoptyses. The patient died on June 17, 1942, and autopsy revealed a hypernephroma of the left kidney with metastasis to the lungs, liver and bone. There was no evidence of pulmonary tuberculosis.

Case V. S. M., male, aged thirty, was admitted to this institution on September 7, 1940, in excellent general condition. The onset of his illness was with cough and expectoration in July, 1939. A roentgenogram taken in May, 1940, showed what appeared to be extensive tuberculosis of both lungs. His sputum was negative. He was treated for tuberculosis in a private institution and because of lack of funds he was admitted to our institution. The roentgen findings could easily be interpreted as being due to tuberculosis (Fig. 5). There was an enlarged, freely movable but painless gland at the right temporomandibular angle, of one year's duration. All sputum examinations were negative for tubercle bacilli. The Mantoux test was negative. A biopsy of the gland revealed the classical picture of Boeck's sarcoid. The roentgen changes were due to this condition rather than to pulmonary tuberculosis.

DISCUSSION

The diagnosis of chronic lung disease is not always an easy matter. The first consideration when abnormal roentgenologic findings exist is usually pulmonary tuber-

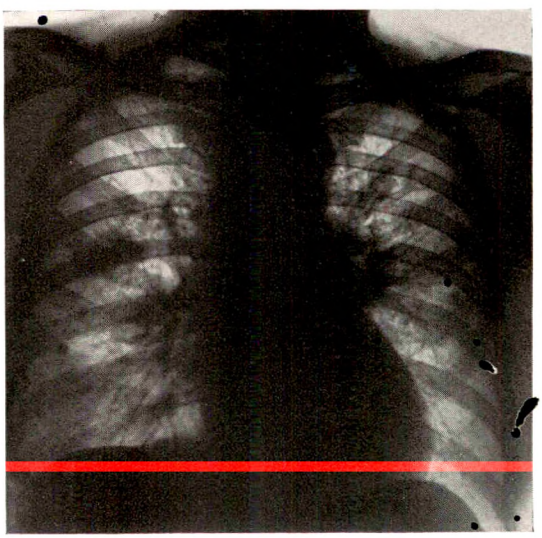


Fig. 4. Case IV. In the right mid-lung field, and to a lesser extent in the base, are shadows giving the roentgen appearance of infiltration and exudation —in the left upper lobe at the second interspace peripherally is an annular configuration. That the original lesion was a cavity in the left lung with a contralateral bronchogenic cross infection could be erroneously concluded from the roentgen findings alone.

culosis. Although the roentgenogram is merely a shadow picture, the pathological entities of tuberculosis, namely infiltration, exudation, caseation, cavitation, fibrosis and the combinations of these, can be ascertained in the roentgenogram. Yet these abnormal pathological roentgen changes can be seen in conditions not produced by tuberculosis. In the first case, of aleukemic lymphadenosis, the roentgenogram presented very fine areas of density extending from the subapical region and becoming heavier above both diaphragms. These dense shadows could easily have been interpreted as being due to a form of hematogenous tuberculosis of the lungs. In this blood dyscrasia there is a withdrawal of lymphocytes from the blood with storing of these cells in tissue spaces, namely the lungs, spleen and liver, as occurred in our patient. The roentgen findings in the lungs are due to lymphatic infiltrations of the peribronchial and interlobar connective tissues. The sternal puncture showed practically all lymphocytes, indicative of metaplasia of the bone marrow, which is pathognomonic of lymphadenosis. The absence of tubercle bacilli in the sputum, and the presence of confirmatory findings of aleukemic lymphadenosis, led to the correct diagnosis.

A case of aleukemic lymphatic leukemia, with lung infiltrations, was reported by Wiseman.[1] In Falconer and Leonard's[2] 30 cases of lymphatic leukemia, 9 showed pulmonary dissseminations.

In the case of calcicosis, the roentgenogram showed massive fine infiltrations from the apex to the diaphragm in the right lung, with similar fine infiltrations in the left lung. Another roentgenogram, taken less than three months after admission, showed a complete disappearance of the massive bilateral infiltrations, leaving no residual deposits. The patient had been inhaling a fine chalk dust, calcium carbonate, used as a "stamping powder" for a period of eleven years in his work as a floor man in an embroidery concern. Knowing the radiopacity of calcium in pulmonary fields, it was felt

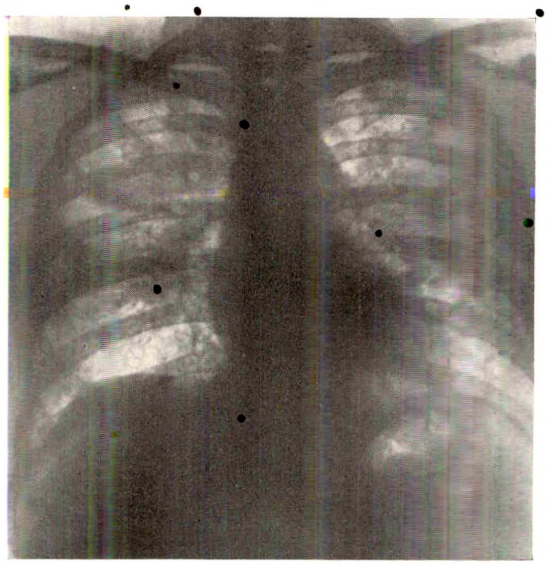


FIG. 5. Case v. There is a fairly symmetrical involvement of both lungs from the second to the fourth ribs anteriorly of the right lung and from the second to the fifth ribs anteriorly of the left lung. The involvement appears to consist of infiltration and collateral inflammation with suggestion of an annular shadow at the third rib anteriorly of the right lung.

that chalk dust and not pulmonary tuberculosis was the cause of the massive pulmonary infiltration. It would be expected that such material would cause clouding of the bronchial tree only, but the possibility of lymphatic spread, with engulfment of the particles by phagocytes in the alveoli, might account for the appearance in this case. Authorities have placed dusts rich in calcium as the least harmful of the inorganic mineral dusts. Calcium is a normal constituent of the body tissues and is soluble in body fluids. It is reasonable to suppose that, chemically, calcium is harmless in the body. Rogers[3] described the roentgen appearance in persons working with marble dust which is essentially calcium carbonate. There were a large number of fine nodules, not only in the hilum, but scattered throughout the lungs, which he interpreted as calcifications. These persons have "marble workers' phthisis," which we feel our patient had, and not tuberculosis.

Adenocarcinoma of the lung, as occurred in our patient, was difficult to diagnose.

The dense shadow limited to the infraclavicular region of the right upper lobe first became manifest in 1935, and could easily be explained on a tuberculous basis. A roentgenogram taken three months later showed no change. Cough and expectoration were minimal, probably because the vesicular tissue of the lung was chiefly involved. Sputum was persistently negative. The toxic goiter was producing his extreme nervousness, cardiac symptoms and the loss of weight of 10 pounds in eight weeks. After the thyroidectomy was performed at the Mayo Clinic, he gained 22 pounds in weight in one month. However, the shadow in the right upper lobe showed slight progression. There were no disturbing pulmonary symptoms. The pneumonectomy performed at the Mayo Clinic revealed a Grade 4 adenocarcinoma of the right upper lobe, with involvement of the regional hilar lymph nodes. No tuberculosis was found. In this case we have a patient with primary carcinoma of the lung, of a number of years' duration, only slightly progressive from a roentgen standpoint. The cough and expectoration had always remained minimal.

Boeck's sarcoid occurs infrequently and usually atypically. The pathological changes may be entirely confined to the lungs and the roentgen appearance may be identical with that of pulmonary tuberculosis to the extent that annular shadows may be seen in the roentgenogram and interpreted as cavities. The cough may be severe and the expectoration profuse. However, tubercle bacilli are absent. The Mantoux test is usually negative. A biopsy of a gland, which does not have to be increased in size, reveals non-caseating tubercles that are pathognomonic of this condition. Skin and bone changes need not necessarily occur in Boeck's sarcoid.

SUMMARY AND CONCLUSIONS

1. Cases of aleukemic lymphadenosis with lung changes, calcicosis of the lungs, adenocarcimona of the lung, hypernephroma of the left kidney with metastasis to the lungs, and Boeck's sarcoid, erroneously diagnosed as pulmonary tuberculosis from roentgenologic findings alone, have been presented.

2. In tuberculosis case finding, the roentgen ray plays a great part in the diagnosis of this disease. However, a plea is made not to be too hasty in making the diagnosis of pulmonary tuberculosis on roentgen findings alone without consideration of other laboratory procedures.

3. It cannot be emphasized too strongly that when a patient expectorates sputum which originates from below the bifurcation of the trachea, that sputum should contain tubercle bacilli if an open case of tuberculosis exists. The failure to find tubercle bacilli in the sputum should result in an investigation for non-tuberculous pulmonary disease as shown in my series of cases.

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CARDIAC MENSURATION*

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THE need for an accurate method of detecting cardiac enlargement is attested by the voluminous literature on the subject, and the many methods described.[2,4–8,10–13] Evaluation of cardiac disability is difficult at best, and cardiologists persist in putting dependence on the roentgenological determination of heart size. This is particularly true in insurance and compensation examinations, and the need for dependability and reasonable accuracy in these cases is obvious. The problem has been attacked by many investigators during the past two decades, culminating in the prediction tables formulated by Ungerleider and Clark,[12] and the nomograms of Ungerleider and Gubner.[13] The majority of these calculations have been based on (1) the transverse diameter, or (2) surface area of the cardiac silhouette. Volumetric methods have been excluded from this discussion deliberately, because they are felt to be too complicated and tedious for routine use.

Comparison of actual cardiac weight at autopsy with the roentgen estimation of cardiac size has convinced us that a constant ratio exists between these two values. It is realized that cardiac "weight" and "size" are not synonymous terms; however, the consistent, positive correlation we have found between weight and size (the latter estimated by roentgen methods) is significant enough in our estimation to warrant the publication of this preliminary report.

Cardiac mensuration is most useful to the clinician for the detection of early cardiac enlargement before dilatation occurs, since it is at this stage that clinical signs are meager and inconclusive. Conversely, a dilated, decompensated heart produces abundant objective evidence of its status, requiring no confirmation by indirect methods. Since any increase of cardiac weight above normal is an absolute indication of cardiac enlargement (and therefore of cardiac disease), this value, if predictable, should be the most accurate yardstick of cardiac size.

A table of normal cardiac weights was recently published by Zeek,[14] based on the study of over 9,000 necropsies, of which only 900-odd cases fulfilled her rigid criteria of normality regarding the cardiovascular system. She concluded that in the normally nourished individual, there is a constant, positive correlation between heart weight and body length, and formulated a prediction table on that basis. Many writers feel that body weight is more significantly correlated to heart weight than is body length, and that both should be used in arriving at a normal value. Rosahn[9] dismisses body length entirely as a significant factor. However, we feel that Zeek's conclusions are well substantiated by her statistical analysis of a large and representative series of cases, and we have, therefore, adopted her table of normal values as our basis of comparison.

MATERIAL

The cases of 200 adult males ranging from twenty-four to seventy-four years of age were selected for study, in which both cardiac weight, as determined at autopsy, and teleroentgenographic measurements, made within the three month period immediately preceding the patient's death, were available. A large percentage of the roentgenograms were actually made within a few weeks ante mortem, but three months was selected as an arbitrary maximum time limit. Twenty-one cases of emaciated or obese individuals and 18 with pericardial effusion were not used in computing aver-

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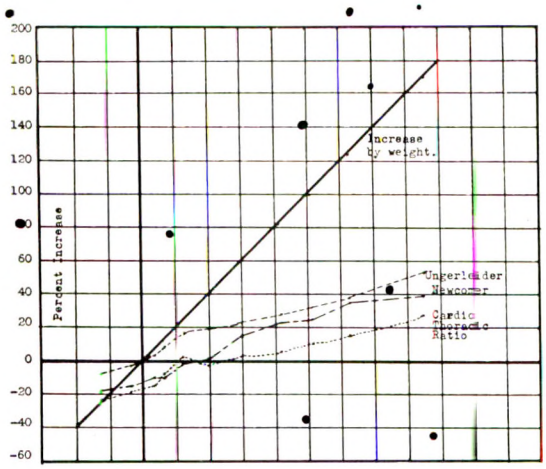


Fig. 1. A graphic expression of the data contained in Table I. The failure of the Newcomer method and cardiothoracic ratio to detect any enlargement less than 40 per cent by weight is shown. It will also be noted that none of the methods indicated enlargement where the cardiac weight was not also abnormally high.

ages because of the known error introduced by these factors. Illustrative cases of this type are presented in Table II. Thus, 161 hearts were actually used in the main analysis.

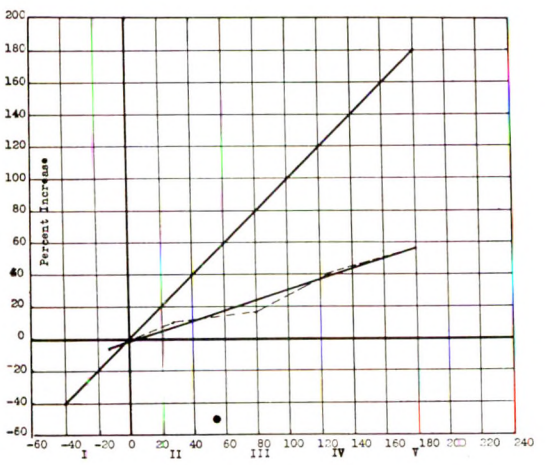


Fig. 2. An "average" comparison between enlargement as shown by weight, and as obtained by the Ungerleider method. The average ratio between the results by the two methods is shown to be of the order of 3.3:1; i.e., a heart that is 30 per cent enlarged by the Ungerleider method could be expected to be approximately 100 per cent enlarged by weight.

Methods

Roentgenological methods chosen were those of Ungerleider,[12,13] *et al.*, Newcomer and Newcomer[8] and the "cardiothoracic ratio." The Ungerleider method was adopted in this hospital soon after its publication, and we have felt that its values were most consistent with clinical findings. It consists simply in the comparison of the transverse diameter, as measured on the teleroentgenogram, with the predicted normal given in their table. The Newcomer method was selected as representative of those utilizing the measurement of surface area. It is based on the ratio of the area of the cardiac rectangle to the area of the thoracic rectangle, the predicted normals being 20, 22, and 26 respectively for patients of asthenic, medium, and sthenic builds. The cardiothoracic ratio was used because, although open to much criticism,[1,5,8] it is still employed in a surprising number of hospitals and clinics. The customary value of 50 was set as the upper limit of

Table I

Average enlargement of the heart expressed in per cent for each of twelve groups, by weight, and by the three roentgen methods.

Group Enlargement by Weight	Average of Group Enlargement by Weight	Average of Group Enlargement Ungerleider	Average of Group Enlargement Newcomer	Average of Group Enlargement Cardiothoracic Ratio
Above 200% (one case)	330	43	95	30
150–200	170	54	39	28
110–150	136	38	35	17
90–110	100	33	24	11
70–90	83	28	22	6
50–70	58	22	15	4
30–50	40	18	0	1
20–30	22	15	−1	−1
10–20	15	6	−10	−9
0–10	4	2	−9	−15
0 to −10	−5	−4	−15	−18
−10 to −30	−24	−6	−18	−24

TABLE II

Representative cases in which there was a marked discrepancy in the results, due to obesity, emaciation and pericardial effusion. In Column 9 is given the corrected Ungerleider value based on the normal rather than the patient's actual weight.

Case No.	Height cm.	Weight lb.	Heart Weight gm.	Transverse Diameter cm.	Predicted Transverse Diameter cm.	Percentage Enlargement Ungerleider	Percentage Enlargement Zeek	Ungerleider Percentage after Weight Correction	Diagnosis
			Overweight						Overweight
22	170	220	600	17.1	15.6	9	87	21	Hypertensive cardiorenal disease
131	172	198	535	14.6	14.7	0	64	15	Coronary occlusion; hypertension
33	165	165	510	13.0	13.7	−5	64	8	Chronic glomerulonephritis
			Emaciated						Emaciated
81	169	99	350	12.5	10.5	19	10	2	Carcinoma of esophagus
162	175	118	365	14.1	11.2	27	10	7	Tuberculosis, pulmonary, chronic
95	171	90	350	15.2	10.0	52	8	13	Pneumoconiosis and bronchiectasis
			Pericardial Effusion						Pericardial Effusion
15	189	170	440	16.6	13.0	27	23		Anemia, myelophthisic, with anasarca (250 cc. of effusion)
53	174	170	405	19.6	13.6	42	23		Hemopericardium due to metastatic tumor (1,000 cc. of effusion)

normal. The assembled data were listed according to increasing magnitude. All measurements, including increase in weight based on Zeek's table of normals, were reduced to percentage deviations.

In depicting our findings graphically, the cases were arbitrarily grouped according to heart weight (see Table 1). The group limits were selected so as to obtain a representative number of cases in each division. The mean percentage increase for each group was calculated for weight, and for the three roentgen methods of measurement. The increase by weight was graphed as a straight line, and the mean for each method of measurement was graphed below the mean of the weight increase for each group. The data thus acquired are presented in Table 1 and Figure 1.

Figure 2 is drawn to show the relationship between the results by the Ungerleider method and the actual percentage increase in heart size, as demonstrated by an increase in weight. The groupings were spaced at intervals of 50 per cent, and the cases fitted into the grouping. The mean of each group was again calculated, and the results graphed. Since all the data consisted of one factor—percentage increase or decrease from normal—the curve was not straightened mathematically, but an average line was selected and graphed.

ANALYSIS OF DATA

1. There is a positive correlation between the percentage enlargement in terms of cardiac weight, and the percentage enlargement as measured by the three roentgen methods used.

2. The values obtained by the Ungerleider method more closely approximate the enlargement by weight than do those by the other two methods, particularly in border line cases. The Ungerleider method is the only one of the three by which enlargement of 40 per cent or less can be detected.

3. As the percentage by weight increases, the disparity between its value and that obtained by the three roentgen methods becomes greater, at a constant ratio, the value of which, as shown in Figure 2, is approximately 3.3 to 1.

4. The results by any method of cardiac mensuration, based upon body weight as a factor, are necessarily influenced by any gross variation in either direction from a normal weight value. Representative cases from our series are illustrated in Table II. It will be noted that the values in obese patients are deceptively normal in the presence of definite cardiac enlargement, and are equally fallacious in the opposite direction in persons exhibiting a marked weight loss.

5. Pericardial effusion of the order of 200 cc., or more, produces a false increase in the transverse diameter by all roentgen methods, as illustrated in Table II. Less than this amount is apparently so thinly distributed within the sac as to have no perceptible effect.

DISCUSSION

Prediction of cardiac size, using body weight as one factor, can result in a considerable degree of error, as indicated in Table II. Zeek has shown that obese and extremely muscular individuals should not be considered as "normal." For example, a male 68 inches tall should weigh 160 pounds,[3] and according to Zeek's table, his heart should weigh 334 grams (±40). In another individual of the same height and weight, but of very muscular build, or of the same height but 40 pounds overweight, the heart might weigh circa 400 grams, and still be within normal limits. When it is realized that a large proportion of cardiac disease is associated with obesity, the problem of the establishment of normal standards of cardiac size becomes apparent. In undernourished patients, we are confronted with the same sort of error, but in the opposite direction; this is illustrated by the second group of cases in Table II. We believe that an arbitrary limit of variation from standard weight should be agreed upon, beyond which the usual prediction tables should not be used unless the standard weight value is substituted for the actual weight. We have tentatively set this limit at ±10 per cent, based on results obtained by trial or error methods in the group of cases excluded from the main analysis because of grossly abnormal weight. The results of this correction are carried in Column 9, Table II.

The clinician may be further deceived by values obtained in serial examinations if there has been an appreciable change in body weight during the interim, unless (1) a constant weight, or (2) the standard or "normal" weight value is used throughout the series.

No method of mensuration can be expected to differentiate between hypertrophy and dilatation; this problem can best be solved by the clinician, based upon his findings and serial teleroentgenograms. In our series, however, a disproportionate increase in the transverse diameter as compared to heart weight was not observed, suggesting that dilatation is a negligible source of error in these calculations. Furthermore, as Comeau and White state so aptly, "Cardiac mensuration . . . should not be accepted unreservedly [as an indication of cardiac disease] . . . one must combat such a tendency as in any other laboratory procedure in which mathematical limits are established." Roentgenoscopy, electrocardiography, and the various functional tests must be employed conjunctively in any comprehensive examination.

The comprehensive analysis of methods

of cardiac measurement made by Comeau and White[1] is predicated on clinical findings. Our study seeks to supplement their work by devising a means of estimating actual cardiac "size" based on transverse cardiac diameter. An obvious defect of any system measuring only one cardiac dimension is the fact that values expressed as percentage deviations from normal may be misinterpreted by clinicians as being expressions of the actual degree of cardiac enlargement, whereas (in the Ungerleider method, for example) the value refers to a deviation in one plane only. After considerable consultation and discussion, we have adopted the following wording of roentgen reports: "Estimated heart size—per cent of the predicted average normal," the percentage being the product of the increase in the transverse diameter above the Ungerleider predicted normal, and the factor 3.3, plus 100. For example, if the transverse diameter is plus 20 per cent of the predicted normal, the report would then read: "Estimated heart size 166 per cent of the predicted average normal."

This preliminary report is presented with the hope that the problem will be investigated by others with particular reference to (1) the reliability of the factor 3.3 arrived at in the present study, and (2) the limits in deviation of body weight from standard weight (for a given age and height), within which a reliable estimation of normal transverse diameter can be predicted on a basis of actual rather than normal or standard weight.

SUMMARY

1. A direct comparison was made between the transverse cardiac diameter of 200 adult males, obtained within ninety days of death, and the weight of their hearts at autopsy.

2. In this study, the results by the Ungerleider method are much more closely correlated with the actual cardiac weight than are those of the other two roentgen methods studied.

3. There is constant correlation between the percentage deviation of the transverse diameter, as obtained by the Ungerleider method, and the percentage deviation in heart weight as calculated from Zeek's table. This correlation expressed numerically has a value of 1 to 3.3.

4. Marked deviation from normal body weight and pericardial effusion greater than 200 cc. impair the accuracy of the method to such an extent as to preclude its use without qualification.

The authors gratefully acknowledge the assistance of David J. Shellenberg, R.T., in the preparation of tables and graphs.

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A SIMPLE GRAPHIC METHOD FOR MEASURING THE AREA OF THE ORTHODIAGRAM*

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ONE of the methods commonly used for estimating the size of the heart is the determination of the area of the frontal projection of its roentgen shadow. This is usually done from the orthodiagram with the superior and inferior borders drawn in conjecturally. The measurement of the actual area contained by the outline is usually accomplished either by means of a planimeter or by transferring the outline to squared-paper and counting the squares within it. The area of squares transected by the outline is estimated. If done accurately, the squared-paper method is tedious. The planimeter while accurate and convenient is not available in all hospitals or physicians' offices. Terrill[6] has described a simple gravimetric method for measuring the area of the cardiac shadow on the roentgenogram and has applied it in 20 cases. A similar method for use with the orthodiagram has been adapted by one of the authors[3] from techniques previously utilized by Nissim[4] and Strong and Gordon.[5] By applying the method in 100 cases it was shown to be sufficiently accurate for clinical purposes. Its main disadvantage is that it requires the use of a chemical balance.

Van Buskirk[7] has described a graphic method for measuring the cardiac area which uses rectangular coordinates. However, he gave no data on the accuracy of the method or its source of error. The method described in this paper is based on essentially the same principle but its application is simpler. Moreover, the use of the method in 100 cases shows it to be quite precise.

METHOD

If a line (*x*-axis in Fig. 1) is drawn through the longest axis of the outline of the frontal projection of the heart shadow, it transects it into two segments. The problem of measuring the area of each segment is that of determining the area under a curve. The length of the base may be measured directly. If the average altitude of each curve were known, the area under it could be computed from the formula for the area of a rectangle, namely, base × altitude. A simple method for readily determining the base and average altitude by means of a transparent grid has been devised.

A rectangle of unexposed, fixed, roentgen film 20×25 cm. is ruled with black india ink into squares by two series of parallel lines 1 cm. apart, perpendicular to each other. This may be conveniently done by fastening the film over a piece of centimeter graph paper and tracing the appropriate lines on the film. The film held with the longer axis horizontal is numbered along the middle horizontal line (or *x*-axis) from 0 to 24 at each point where it is intersected by the vertical lines. The horizontal lines are numbered along the left hand margin (or *y*-axis) in both directions from the middle horizontal line, which is numbered 0. The centimeter intervals between the parallel horizontal and vertical lines are then ruled with 4 lines to make intervals of 0.2 cm. This is best done with red ink. A diagram of a portion of the completed grid is shown in Figure 1. The intervening 2 mm. spaced lines are not shown in the diagram.

The use of the grid is as follows: A pencil line is drawn along the longest axis of the orthodiagraphic tracing (*x*-axis in Fig. 1). If properly drawn, lines perpendicular to it where it intersects the heart outline should be tangential to the outline. The grid is placed upon the orthodiagraphic tracing so

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that its *x*-axis or zero line coincides with the long axis of the outline and with its *y*-axis tangential to the left edge of the outline. This is shown in Figure 1.

The outline of the heart shadow is inspected through the transparent grid. At each point at which it intersects the black vertical lines the distance along the *y*-axis, estimated to the closest millimeter, is noted. This is done both above and below the *x*-axis. The values are all added, the minus signs being neglected. The sum of the values is divided by the number of vertical lines transected. This can be read directly from the grid along the *x*-axis. The result of the division represents the sum of the average altitude above and below the *x*-axis. The length of the base (*x*-axis in Fig. 1) to the nearest millimeter is read from the grid. This value multiplied by the altitude gives the area of the cardiac outline in square centimeters.

The computation is illustrated by the following example: In one case the sum of the readings at each point at which the vertical lines were transected by the outline was 145.4 cm. The number of vertical lines transected was 15. Each vertical line was, of course, transected twice, once above and once below the middle horizontal line (*x*-axis). The length of the base was 15.2 cm. The area, therefore was

$$\frac{145.4}{15} \times 15.2 = 147.3 \text{ sq. cm.}$$

The method as described is essentially a simplified application of a theorem in approximation calculus. The theorem may be stated: "The average value *y* of a function for an interval equals the average of the average values for *n* equal sub-intervals."[1]

APPLICATION

The method as described was applied to the measurement of the area of 100 orthodiagrams and arbitrarily drawn figures of the shape and size of the heart. Each determination was compared to the average of three planimeter readings on the same figure. The average deviation from the planimeter determination was 0.73 per cent. Thirty-nine of the determinations showed a negative error with an average of 0.85 per cent and 61 a positive error with an average of 0.75 per cent. The largest

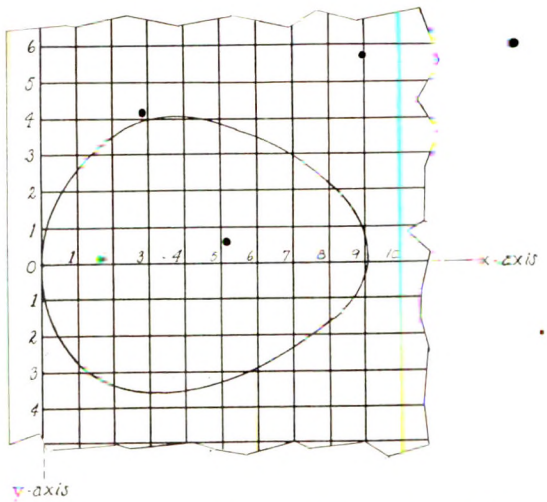


FIG. 1. Diagram of grid superimposed on cardiac outline. The 0.2 cm. spaced lines are omitted for clarity. In actual practice the cardiac area is larger in proportion to the size of the grid than shown.

negative error was 2.3 per cent, the largest positive error 3.6 per cent.

The determinations were all made by one individual. After the readings were taken the grid was removed, reapplied, and the readings checked. In a series in which the readings were not checked in this manner the deviations from the planimeter readings were slightly higher.

SOURCES OF ERROR

There are four main sources of error in the application of the method.

(*a*) *Errors in the Grid.* These may be eliminated by careful measurements of the completed grid.

(*b*) *Improper Drawing of the Long Axis of the Orthodiagram.* This may be checked by drawing tangents to the figure where it is intersected by the long axis. The tangents should be perpendicular to the long axis if the latter is properly drawn.

(*c*) *Improper Placing of the Grid.* This is

the most important source of error. Particular care must be taken in placing the *y*-axis of the grid at the left hand edge of the figure. The tangent drawn to check the position of the long axis will be of assistance in doing this accurately. To avoid movement of the grid and figure during the measurements, they should be fastened to a flat surface with thumb tacks.

(*d*) *Errors in Readings.* The actual measurements are made to the nearest millimeter. Since, as a rule, more than 20 such readings are averaged in determining the altitude, a small error in one will not be of great significance. Most important is the final measurement along the *x*-axis. Since the average altitude is multiplied by this value for the final result, errors in its measurement will be reflected there directly.

SIMPSON'S METHOD

The possibility of applying a simplified version of Simpson's rule[2] to the problem of determining the area of the orthodiagram was also investigated. Simpson's rule gives a close approximation of the average height of curves of first, second, or third degree. In applying the method to 80 orthodiagrams or arbitrarily drawn areas of the size and shape of the heart, the average deviation from the planimeter reading was 2.8 per cent. In 7 instances the deviation was greater than 5 per cent; the greatest error was 12.4 per cent. Since the method is more time consuming and considerably less accurate than the technique already described, the details of its application are not given.

SUMMARY

A simple graphic method for the determination of the area of the orthodiagram is described. It requires no equipment not readily available to any physician. Its application to 100 cases checked by the planimeter shows it to have a sufficiently high degree of accuracy for clinical purposes.

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ACUTE SUPPURATIVE ARTHRITIS OF THE HIP IN CHILDHOOD

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ACUTE suppurative arthritis of the hip continues to be the cause of many grave deformities, so that serious consideration must be given to it by those responsible for the diagnosis and treatment of this disease.

Since the greatest incidence is in children from early infancy up to the age of fifteen, our cases were selected entirely from that age group. Cases of acute suppurative arthritis due to deep penetrating wounds or severe crushing injuries of the hip were purposely omitted.

In the discussion of acute suppurative arthritis we shall consider: (1) the anatomical factors; (2) types of arthritis; (3) bacterial characteristics of the infection, and (4) roentgenological aspects. Under anatomical factors are included the bones and capsule of the hip joint, its blood supply and lymph drainage.

1. *Anatomical Factors.* The adult acetabulum is formed by fusion of portions of the iliac, ischial and pubic bones. Fusion of these bones occurs approximately at about the age of seventeen.[6] Prior to this age these three bones are united by a Y-shaped piece of cartilage. The capital epiphysis of the femur begins to ossify in the later six months of the first year of life, and fuses with the femoral neck at approximately the age of seventeen. The acetabulum, femoral head, and the greater portion of the femoral neck are intracapsular in location. Since the highest incidence of septic infections of the hip is found in the period of early childhood and adolescence, it is obvious that the hip joint is most vulnerable to infections during this period of active growth and before fusion of the epiphyses has taken place. How important a rôle this growth factor plays is rather difficult to evaluate accurately.

A discussion of the blood supply of the hip presents several difficulties: First an accurate knowledge of the blood supply to this joint is incomplete. Second, anastomosis between the various arteries does not occur until after the eighth year. Third, since this infection is usually of hematogenous origin it may attack one or more tissues of the hip joint.

Through the excellent work of Wolcott[12] and of Chandler and Kreuscher[3] the existence of blood vessels within the ligamentum teres has been confirmed. These vessels carry blood to the femoral head; however, it is acknowledged that these blood vessels are variable in size and number and in certain cases may be totally absent. It is noteworthy to mention at this point that the femoral epiphysis is rarely if ever the primary site of an intra-osseous lesion. The main blood supply to the hip is by branches of the capsular and nutrient arteries. The capsular branches after piercing the capsule of the joint enter the neck of the femur through multiple foramina located on the posterior superior surface of the neck. Thus in the femoral neck an anastomosis is established between the capsular branches and branches of the nutrient artery. According to Logròscino[8] the branches of the nutrient artery are end arteries until a child reaches the age of eight, and anasto-

TABLE I

AGE INCIDENCE

	Cases Reported	1 to 15 Years	16 Years and Over
Eadgley, *et al.*[1]	113	99	14
Caldwell[2]	17	17	
Slowick[11]	60	39	21
Our series	40	40	

mosis takes place after this period. In our series the greatest incidence of acute suppurative arthritis increases appreciably in children past the eighth year. In 40 children all under fifteen years, acute suppurative arthritis is reported in 13 cases in children under nine years and 27 cases in children over nine years. The ninth year was chosen as an arbritary year because none of these children was found to be in that age group.

Most of the lymphatic drainage of the hip is to a chain of lymph nodes located about the external iliac artery, except the posterior portion of the hip which drains to glands about the hypogastric artery. Inguinal, femoral and Cloquet's glands also have efferent vessels to the external iliac nodes. The external iliac and hypogastric nodes drain into a common group of glands situated about the common iliac artery and are presacral in location. The external iliac nodes are retroperitoneal and lie immediately above the fascia of the iliopsoas muscle. Thus suppuration and abscess formation in any of these variously located glands, burrowing along fascial planes, may explain the confusing clinical manifestations often encountered in these cases. Freiberg and Perlman[5] recently reported several cases of acute suppurative arthritis with either a pelvic or abdominal abscess which were operated upon as cases of appendiceal abscesses. These authors stress the importance of the lymph drainage of the hip and describe it in detail to illustrate the various clinical pictures that a case of septic hip may present.

2. *Types of Acute Suppurative Arthritis.* Phemister has classified acute suppurative arthritis of the hip into the primary and secondary type depending on which tissues of the hip are involved. In the primary type the infection attacks the synovial or perisynovial structures, whereas in the secondary type the infection is primarily intraosseous in location, and, due to spread or extension of the bone infection, the joint is secondarily affected. There are several points of difference between the two types. The primary type is in most instances due to a streptococcal infection, the disease usually subsides within five weeks, and the affected hip in many cases regains its normal range of motion resulting in very little if any deformity of the hip. On the other hand, the secondary type is usually caused by staphylococcal infection, runs a long drawn out course, draining sinuses taking many months or years to heal, range of motion is in most cases markedly impaired, ankylosis and deformities of the hip result in the majority of cases. In our series of 40 cases, 5 cases, or 12 per cent, are of the primary type, and 37 cases, or 64 per cent, are of the secondary type. Although these percentages are not an accurate indication of the frequency with which the two types occur, we do know that the primary type is more common in very young children, and the secondary type more common in the older child. Since the incidence of septic hip is greater in the older child it follows that there are more cases of the secondary type.

3. *Bacterial Characteristics of the Infection.* The predominant organisms found to be responsible for acute suppurative arthritis of the hip are the staphylococcus and streptococcus. Although other pyogenic organisms are occasionally found, the above fact has been substantiated by aspiration smears and blood cultures. Another well known fact is that pre-existing infections in other parts of the body such as osteomyelitis, pneumonia, otitis media, erysipelas, and furuncles are predisposing factors to hip infections. Another predisposing factor is injury to the hip. As previously mentioned, the streptococcus usually attacks the synovial or perisynovial tissue and produces a primary type of arthritis. The staphylococcus primarily infects the bone and by spread or extension into the joint produces the secondary type. Slowick in his 60 cases listed 23 cases as of the primary type and the streptococcus was isolated in each instance. In the 5 cases of primary type of arthritis reported herein, there is 1 case due to streptococcus, 1 case to pneumococcus, and 1 case to a mixed infection in which a gram-positive diplo-

coccus and bacillus was isolated. In the remaining 2 cases aspiration smears and blood cultures were reported as negative. In the secondary type, of which there were 35 cases, the staphylococcus was isolated in 12 cases, a mixed infection of streptococcus and staphylococcus in 1 case, and a gram-positive bacillus in 1 case. In 1 case the aspiration smear and blood culture were reported negative, and in the remaining cases no bacterial studies were done because incision and drainage of the hip had been performed before the patient was transferred to our hospital.

In the entire series of 40 cases there was 1 death and that occurred in a child seven months old. This patient was admitted because of an acute suppurative otitis media and later developed infection of the hip joint. On aspiration and blood culture the pneumococcus was isolated, and several days later when meningitis developed the same organism was cultured from the cerebrospinal fluid. Death occurred on the twenty-third day of observation.

4. *Roentgenological Aspects.* Much of our present knowledge of the various infections of the hip can be directly accredited to the roentgenographic examinations and studies. Roentgenograms are invaluable in detecting the presence of the disease, in following the different phases it goes through, and in evaluating the end-results. However, it has not by any means solved all of our problems in making a differential diagnosis. Even today many clinical and laboratory findings such as blood counts and cultures, aspiration smears, sedimentation rates, and tuberculin skin tests are most useful and are employed together with the roentgen findings in making a differential diagnosis. Phemister[10] in 1924 was the first to demonstrate the tryptic action of non-tuberculous exudate, and that the tryptic action was responsible for the early destruction of articular cartilage, especially cartilage of adjoining surfaces. This early pathology can be detected on roentgenograms by a narrowing of the joint space as early as five to ten days after onset of the disease. Chont[7] in a recent article describes another early roentgen finding in children under two years of age: a lateral and upward subluxation of the femoral head. We would like to point out, however that this subluxation differs from the findings encountered in congenital dislocation, by the associated soft tissue swelling of the pelvic girdle and a ground glass appearance of the bone, which is usually of normal and uniform density in congenital dislocation. In very early cases of hip infection the epiphyses of the femoral heads are equally well developed but differ in density as seen on roentgenograms. In congenital dislocations the femoral epiphysis of the affected side may be absent or is frequently found to be smaller in size. Intra-osseous lesions may not be found or identified on roentgenograms taken ten to fifteen days after onset. After this initial period during which roentgen findings may be reported as being negative, other roentgenograms taken a few days later will show the bone infection and its location. One can anticipate the spread of the infection into the joint in most cases. In these 40 cases the first roentgenogram was reported as being negative in 6 cases, whereas a roentgenogram taken one to two weeks later clearly demonstrated the pathologic changes in the hip. Spread of the infection to other bones of the hip, complications such as pathological dislocation and sequestration and absorption of the femoral head can easily be identified in follow-up roentgenograms taken at suitable intervals. Pathological dislocation which is attributed to rupture of the inferior medial portion of the capsule may occur as early as the first week after onset of the disease. Sequestration of the femoral head and later absorption is a frequent finding. Phemister believes that this is due to rupture of the ligamentum teres thus impairing the circulation to the femoral head. Nicholson[9] in his 113 cases reported that pathological dislocation took place in 34 cases and femoral sequestration in 43 cases. In our series of 40 cases there were 9 cases of pathological dislocation and 6 cases of se-

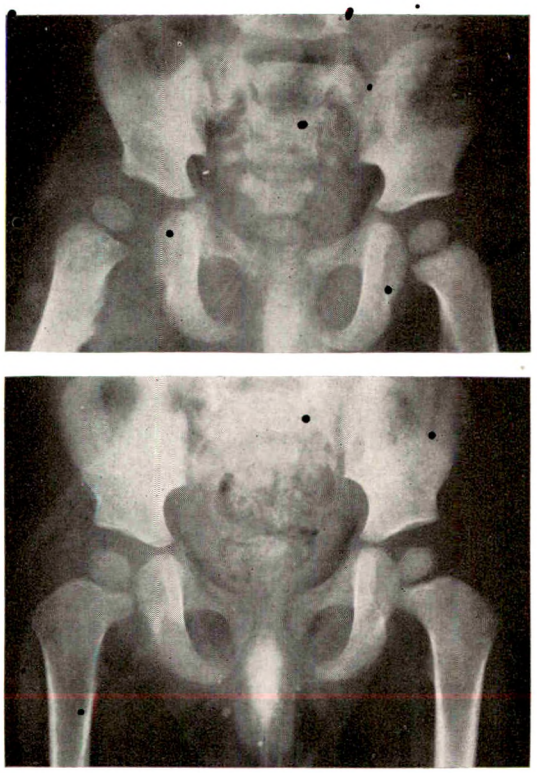


Fig. 1. Case 1. Early case of a primary type of suppurative arthritis in a child sixteen months old, demonstrating the upward and lateral displacement of the right femoral head. Lower roentgenogram shows reduction of the subluxation. (Courtesy of Dr. Chont.)

questration of the femoral head. In 2 cases both conditions were reported. The sequestrated head in most instances is either absorbed or extruded through a draining sinus. Whenever one of these complications occurs one can safely prognosticate a poor end-result, usually a hip with markedly limited motion or even ankylosis. All authors agree that early diagnosis, drainage and immobilization is the treatment of choice and best also for prevention of the above mentioned complications. Whether these complications will be prevented by the recent introduction of the sulfonamides as an important agent in the therapeutic armamentarium of suppurative arthritis will depend on observations made on large material over a sufficient length of time. At present the benefit from the use of these drugs in the early stages seems to be well established.

Finally we see the end-results in these cases. In the primary type the chances of obtaining a nearly normal hip with fairly good motion are better than in the secondary type, but even in this type poor results are sometimes encountered.

Caldwell in his report of 17 cases had 6 cases of the primary type. One of these patients died but in the remaining 5 the hip motion was recorded as being good or normal after the infection had subsided. In this series of 40 cases good or normal hip motion is reported in 7 cases which includes 3 cases of the primary type and 4 cases of the secondary type.

Primary Type—Arthritis

Case 1 (No. 100–599). This patient, a white baby, aged sixteen months, was brought to Crippled Children's Hospital because of a high fever and refusal to move his right leg. The parents stated that five days prior to admission the child refused to stand on his feet or walk and that he became feverish. Examination revealed that the area around the right hip was reddish and swollen and markedly tender. The right hip was held in flexion and any movement caused the child considerable pain. Temperature was recorded as 104° F.; leukocyte count 23,000. A diagnosis of acute suppurative arthritis of the hip was made on admission on March 2, 1939.

A roentgenogram taken on the same day showed a widening of the joint space of the right hip with upward and lateral displacement of the femoral epiphysis. On the day following

Table II

End-results in acute suppurative arthritis of the hip

		Hip Motion Recorded As:		
		Good or Normal	Fair	Poor or Ankylosis
Badgley, *et al.*[1]	113 cases	7	23	83
Caldwell[2]	17 cases	8	5	4
Slowick[11]	60 cases	11	20	30
Our series	40 cases	7	7	23

admission a posterior arthrotomy was performed and on entering the hip joint about 1½ oz. of pus was obtained. Smears and cultures of this exudate were reported as showing a gram-positive diplococcus and bacillus.

The patient's pelvis and lower extremities were immobilized in a Bradford frame and sulfanilamide therapy instituted for twelve days.

The patient was discharged on April 13, 1939, in an abduction traction splint and a notation

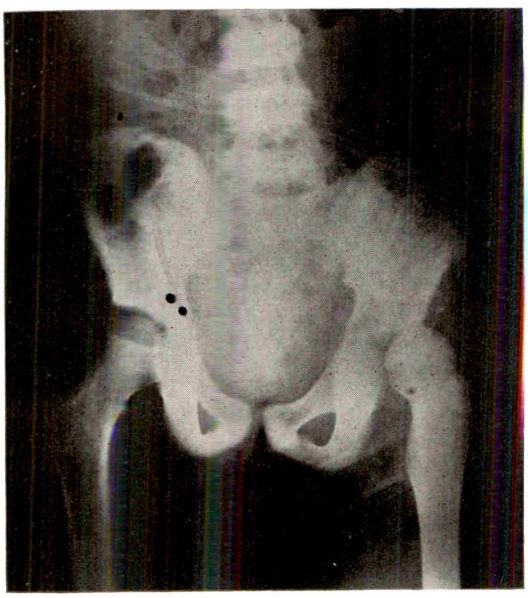


FIG. 2. Case II. Secondary type of suppurative arthritis in a boy eleven years old. The intra-osseous infection has spread from the acetabulum to the ilium and neck of the femur. There is surrounding soft tissue shadow and pathological dislocation of the left femoral head.

indicated that the hip had normal usage of motion, free of pain.

Follow-up roentgenograms taken at intervals of several months, however, revealed a subluxation of the femoral head, and several attempts at manipulative reduction were made. When last seen on April 21, 1941, the child had normal range of motion of the right hip but the right femoral head remained subluxated causing ½ in. shortening of the right leg.

Secondary Type—Arthritis

CASE II (No. 102-872). A white boy, aged eleven, was admitted to the Crippled Children's Hospital on August 27, 1939, because of

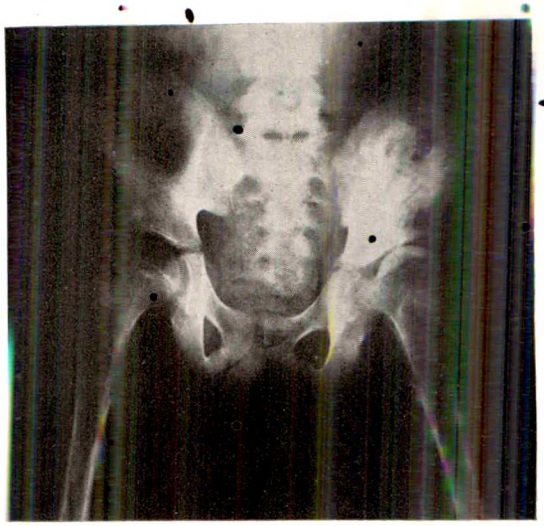


FIG. 3. Case II. Roentgenogram taken five months later showing sequestration and partial absorption of the left femoral head. Several sequestra are seen in the wing of the ilium.

severe pain and swelling of the left hip; the patient appeared to be very toxic and had a high fever. Ten days prior to admission the left hip began to hurt, and at that time the boy had two furuncles on the outer aspect of the hip, but he was taken to the local doctor to



FIG. 4. Case II. Roentgenogram taken two years later showing complete absorption of the left femoral head, partial regeneration of the wing of the ilium following sequestrectomy and ankylosis of the left hip joint.

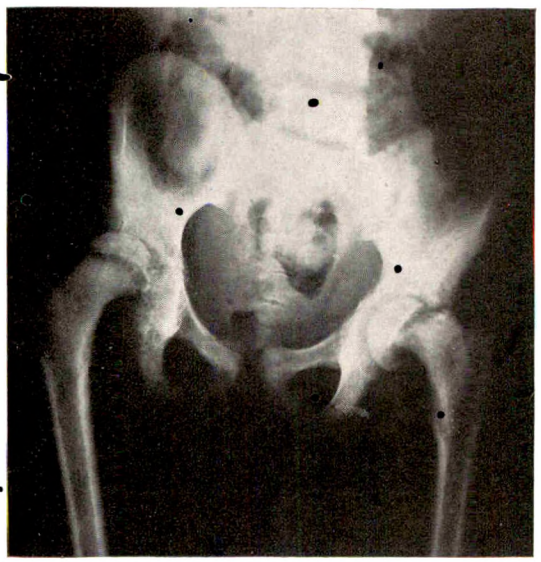


Fig. 5. Another case of secondary type of suppurative arthritis, bilateral, in a girl eleven years old, demonstrating narrowing of the joint spaces, sequestration of the left femoral head and subluxation of the neck of the femur. The right hip shows pathological dislocation.

have a dislocated shoulder reduced. The pain in the hip was attributed to a kick another boy had administered over that area. Five days before admission a fever developed and the boy became very toxic. On admission the left hip was held in marked flexion, the surrounding area was swollen, tender and reddish. The boy was irrational and had a marked elevation of temperature.

A diagnosis of acute suppurative arthritis was made. Aspiration of peri-articular tissues was negative but when the needle entered the joint space considerable purulent exudate was obtained. Aspiration smear and culture were both reported as showing *Staphylococcus albus* as the causative organism. A roentgenogram of the pelvis taken on admission showed no evidence of bone or joint pathologic changes of either hip.

On the day after admission supportive measures were instituted, the left hip was drained through a posterior arthrotomy and chemotherapy instituted. The lower extremities and pelvis were immobilized. At intervals sulfapyridine was discontinued because of renal irritation.

The next roentgenographic examination revealed an osteomyelitis of the acetabulum which later spread to involve the lower two-thirds of the left ilium. Somewhat later subluxation of the femoral head occurred which eventually sequestrated.

This patient has been admitted on several other occasions for the past three years, for manipulations of the left hip—removal of several sequestra in the ilium, and for acute exacerbations of his infectious process in the left hip. When last examined on October 5, 1942, he still had several draining sinuses, and complete ankylosis of the left hip. The boy had spent most of his time since his hip infection in bed and in double hip spica casts.

COMMENT

Acute suppurative arthritis of the hip is a relatively frequent disease of childhood and early adolescence. It occurs in both sexes with equal frequency. In this series of 40 cases there were 22 males and 18 females. Age, on the other hand, is an important factor in that the largest number of cases were found to occur in children ten to fourteen years old. In this age group of patients there were 27 cases, whereas in children under that age there were 13 cases. Although it was not the main purpose of this report to study any racial difference we have only 2 cases occurring in colored

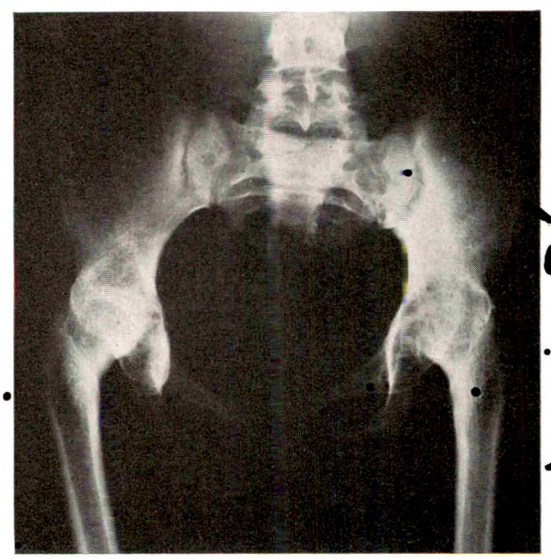


Fig. 6. Same case as Figure 5. End-result shows complete bony ankylosis of both hips.

children. In looking over the hospital records in a total of 142 cases of acute suppurative arthritis of the hip, there were only 12 cases reported as occurring in the Negro. In view of Lewis'[7] observations regarding racial differences as an important factor determining resistance to infections, it may be of interest to note the relatively small incidence of suppurative arthritis in colored patients in our series. Lewis states that an outstanding feature of Negroes as surgical patients is their resistance to infections, especially to suppurative cocci towards which they seem to have a high degree of immunity. This relative immunity is also a part of their resistance to scarlet fever, erysipelas, measles, and certain skin diseases. It is difficult to show any definite conclusions on this point from our series of cases, because our colored wards have much fewer beds than our wards for white children.

In the primary type of suppurative arthritis of the hip affecting chiefly the synovial or perisynovial tissues, and destroying the articular cartilage, the infectious process in most instances can be arrested before any bony involvement develops. This, however, depends on the virulence of the pathogenic organism. If the infection can be brought under control within a short period of time, causing little or no bone destruction, the hip joint will regain its normal range of motion. The primary type is found in very young children under two years of age.

An osteomyelitis of any of the bones of the pelvic girdle with extension into the hip joint is responsible for the secondary type of arthritis, which is by far the more frequent type. The hip joint differs in this respect from the other joints of the extremities in that the epiphyseal line in these other joints usually limits the infection before extension into the joint occurs. It may well be that the anatomical characteristics of the vascular supply and lymph drainage of the hip are responsible for this difference in reaction to an infection. It has been previously stated that the branches of the nutrient arteries supplying the femoral neck are in early life end arteries in the sense of Cohnheim; only later is anastomosis established. This undoubtedly must be of great significance in the presence of a pyogenic embolus in cases of blood borne infections. In the secondary type the articular cartilage is completely destroyed, and the subcartilaginous bone eroded. This process invariably affects the adjoining bones so that multiple bony involvement occurs, and is responsible for the impaired motion of the affected hip, or ankylosis.

TABLE III

ANALYSIS OF 40 CASES OF ACUTE SUPPURATIVE ARTHRITIS OF THE HIP IN CHILDREN UNDER FIFTEEN YEARS OF AGE

AGE: Up to 9 years, 13 cases; from 10 to 15 years, 27 cases

SEX: Males 22; females 18

RACE: White 38; colored 2

PRE-DISPOSING FACTOR: 18 cases: hip infection was preceded by pre-existing infection in some other part of the body; history of injury in 7 cases

TYPES: A. Primary type 5 cases, or 12%
B. Secondary type 35 cases, or 88%
with single intra-osseous lesion of the hip 25 cases located in femoral neck 14; ilium 8, ischium 2, pubis 1 with multiple intra-osseous lesions 10 cases

PATHOGENIC ORGANISM: in 29 cases with fairly recent onset

A. Primary type	1 pneumococcus
	1 streptococcus
	1 gram-positive diplococcus and bacillus
B. Secondary type	12 staphylococcus
	1 staphylococcus and streptococcus
	1 gram-positive bacillus
	4 aspiration smear reported negative
	9 cases no report

COMPLICATIONS: 1. Pathological dislocation, 9 cases
2. Femoral head sequestration, 6 cases
Both complications in 2 cases

END-RESULTS: 40 cases: 2 recent cases: 1 death: total 37 hips

Good or normal motion	7 cases
Fair or 50% motion	7 cases
Poor motion or ankylosis	23 cases

Average follow-up period in 37 cases 3.1 years

CONCLUSIONS

1. The highest incidence of acute suppurative arthritis of the hip in children under fifteen years of age was found in children between the ages of ten to fourteen years.

2. The primary type of arthritis occurs mostly in children under two years of age; above that age the secondary type is by far the more common.

3. In the secondary type of arthritis the most frequent site of the intra-osseous lesion is in the femoral neck, and in the ilium, ischium and pubis in the order mentioned.

4. The great majority of these hips which have recovered from an infectious process show marked impairment of motion or ankylosis.

I wish to thank Dr. A. J. Ackermann for his help and advice in preparing this paper.

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The wrist joint is part of a living dynamic organism and is not a fixed immovable object. Thus, in one set of circumstances, the lines of force may follow the main course indicated on the diagram; at another time, they may take a variant course in the radial axis. There are, of course, modifications of the main stream of force—lateral, torsional, etc. The possible variations of flow of force along the ulnar axis are much less than those along the radial axis. The disabilities of the radial axis are consequently much more numerous and important than the disabilities of the ulnar axis.

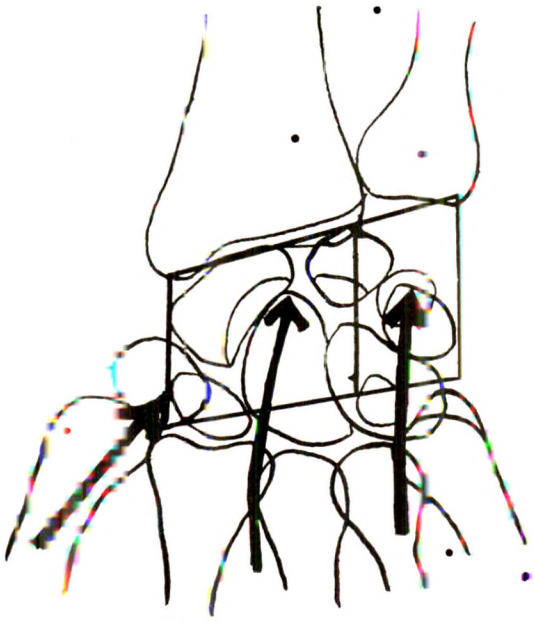


Fig. 2. A distinction is made in this figure of those forces which flow proximally along the most radial part of the radial axis (that is, along the axis of the first metacarpal bone) and the ulnar part of the radial axis. The operative area of carpal traumata is boxed out by the large parallelogram, but it is within the radial parallelogram that most of the carpal traumata take place. The cuneiform bone, and semilunar too, it is seen, occupy an intermediate position in part, so that they are not affected by the full force of trauma applied to either axis.

This axial concept becomes useful for it transcends the individual fracture and joins these various fractures into a whole.

Axial trauma expends itself at the site of fracture, a break occurring at a point in bone where the proximally radiating trauma exceeds bony trabecular and cortical strength. The form of fracture which results depends on (1) the strength of original trauma, (2) its duration, (3) its direction and modification of its direction, (4) the cushioning elasticity or "give" in the joints through which the force is transmitted, (5) the architecture of the bone, and (6) the sudden expenditure of force by break of bone at a point in bone less resistant than the force.

The most direct line for transmission of force of indirect trauma along the radial axis is in a direction parallel to the long axis of the second and third metcarpal bones. The diagram (Fig. 2) illustrates the operative area of carpal traumata. The entire force of trauma is borne by the parallelogram in which os magnum, scaphoid and lesser multangular lie. Only a portion of the greater multangular and semilunar bones lie in the direct projection of the lines of force. The greater multangular enjoys special consideration, for it is in direct line with the long axis of the first metacarpal bone. Fracture or dislocation of the lesser multangular is extremely rare, for this bone is well protected. The semilunar is in a most favorable position since it is unaffected by the full strength of any force which flows along either the radial or ulnar axis. This may explain the rarity of semilunar fractures and the greater relative incidence of rotational dislocation of this bone.

Transmission of force along the *radial part of the radial axis* may give rise in succeeding order to a Bennett fracture,* radial dislocation of the base of the first metacarpal, fracture or rotation of the greater multangular or both, fracture or sometimes rotation of the scaphoid, fracture of the lower end of the radius, and with more proximal transmission, especially with the elbow extended, to disability of elbow or shoulder. When the force is transmitted

* An axial injury to the distal part of the proximal phalanx of the bent thumb, as in boxing, may cause acute or chronic strain of its metacarpophalangeal joint, the force of the blow expending itself at this joint.

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FRACTURES OF THE RADIAL AND ULNAR AXES*

A UNIFYING CONCEPT, WITH A DESCRIPTION OF CERTAIN CARPAL INJURIES, INCLUDING PARALLEL AND GEAR ROTATIONS OF THE CARPAL BONES

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I. FRACTURES OF THE RADIAL AND ULNAR AXES: A UNIFYING CONCEPT

THE mechanism of force, creating a fracture of hand, wrist or forearm, was studied by the clinical history and by roentgenography. The clinical history was frequently incomplete or vague regarding type of trauma; the roentgenogram, however, usually indicated the effect of a trauma to the skeleton. The form of fracture revealed in the roentgenogram is a good clue to the direction of force which made such fracture.

Trauma is direct or indirect. Direct trauma is self-explanatory. In indirect trauma, a force applied distally flows proximally until the flow of force is dissipated by a break of bone. A reverse or distally directed flow does not ordinarily take place, since a direct force applied to the lower arm, or bent elbow, will usually expend itself there.

This indirect or fleeing trauma *with proximal centrifugality* behaves in a characteristic manner, its flow being transmitted along two axes, which we call *the radial axis and the ulnar axis*, respectively. *The radial axis*, in distal to proximal order, includes the first three metacarpal bones, the greater and lesser multangular bones,† the os magnum, the scaphoid, the semilunar, and the radius. The ulnar axis includes the fourth and fifth metacarpal bones, the hamate, cuneiform and pisiform bones, and the ulna. The accompanying diagram shows the constituent elements of each axis (Fig. 1).

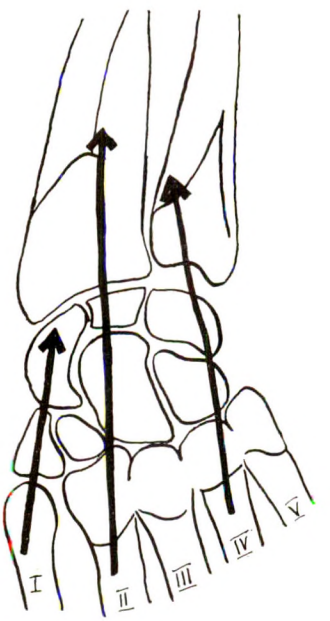


FIG. 1. Diagrammatic sketch showing the proximal transmission of distally applied forces, and the radial and ulnar axes along which these forces flow.

† Where more than one name exists for a carpal bone, use has been made of the more popular. The name starred in the tabulated nomenclature for the carpal bones is used in the text.

*scaphoid	navicular
trapezium	*greater multangular
trapezoid	*lesser multangular
*semilunar	lunate
*cuneiform (triquetrum)	triangular
*os magnum	capitate
*pisiform	pisiform
uniciform	*hamate

* From the Hospital for Joint Diseases, Department of Roentgenology, Dr. Maurice Pomeranz, Director, and the Orthopedic Service of Dr. Harry D. Sonnenschein; and the New York Dispensary.

along the *ulnar part of the radial axis*, these disabilities may arise in succeeding order—fracture of the base of a metacarpal, delayed painful dorsal bony prominence of a metacarpal base, injury of the base of the os magnum, fracture of the neck of the os magnum or dorsal dislocation of its caput, wedge action of the os magnum with resultant mid-carpal dislocation or dislocation of the semilunar, and fracture of the lower end of the radius. Fracture of the semilunar is not common, but we have observed at least two instances of it.

It is not possible to predict accurately the type of injury which may follow axial trauma. Response to such trauma is very different in children than in adults. The tendency in both is for the proximal transmission of trauma along the radial axis since the bearing surface which receives the trauma is usually the ball of the hand. In children, both bones of the forearm or the radius alone is usually broken, the usual site of fracture being at the junction of the lower and middle thirds of the forearm. Epiphyseal dislocation of the lower radial epiphysis is the child's equivalent of the Colles fracture. Fracture of the ulna is quite uncommon as is fracture of the carpus. The axial injury may cause fracture of the head of the radius, the more distal parts being uncamaged.

In the adult, the typical injury is the Colles fracture, since the fracture, which is a fracture through a waist of bone, occurs at a point where the expanded radius narrows. Carpal injuries, especially of the scaphoid, are not uncommon. Injuries of the proximal carpal bones are much more frequent than injuries of the distal carpal row, since the proximal carpal bones are squeezed as in a vise by the axial trauma. There is sometimes association of fracture of various parts of the radial axis, the most common being the association of Colles fracture and fracture of the scaphoid.

Fractures of the ulnar axis are less common. Fractures of the hamate, cuneiform, or pisiform bones are simple fractures, as a rule, whose causative trauma is usually direct rather than indirect. They are seldom associated with radial axis injuries. The cushion of the discus articularis most probably protects the ulna against fracture by indirect trauma but such fracture may result from a fall on the ulnarly directed hand. The lower end of the ulna may be fractured by direct trauma to the palmarly

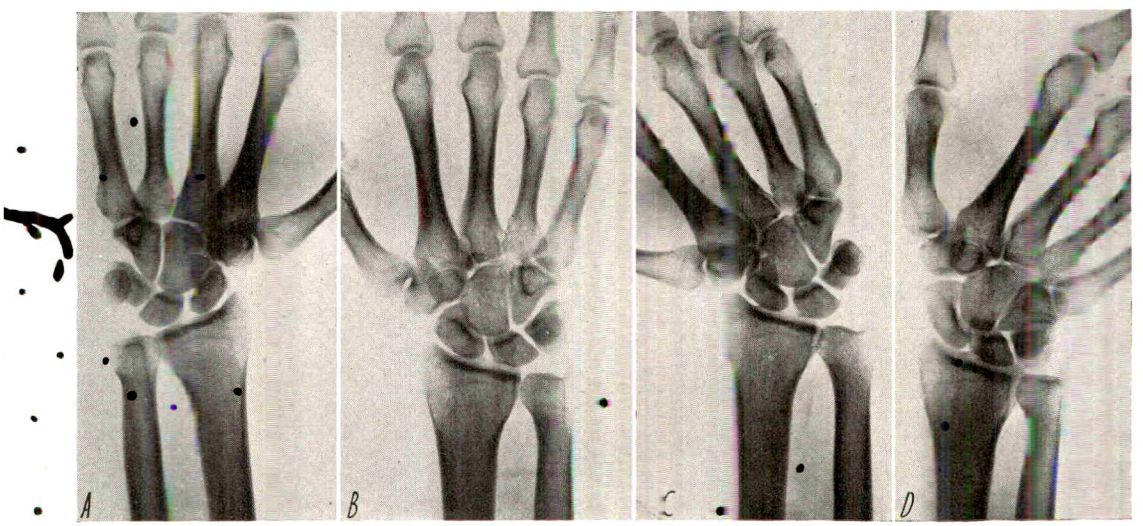


FIG. 3, *A–N*. The roentgenography of the normal wrist. Various views of the wrist were taken, and these roentgenograms are added for a clearer understanding of the text.

A, anteroposterior view; *B*, posteroanterior view; *C* posteroanterior view in radial abduction; *D*, posteroanterior view in ulnar adduction.

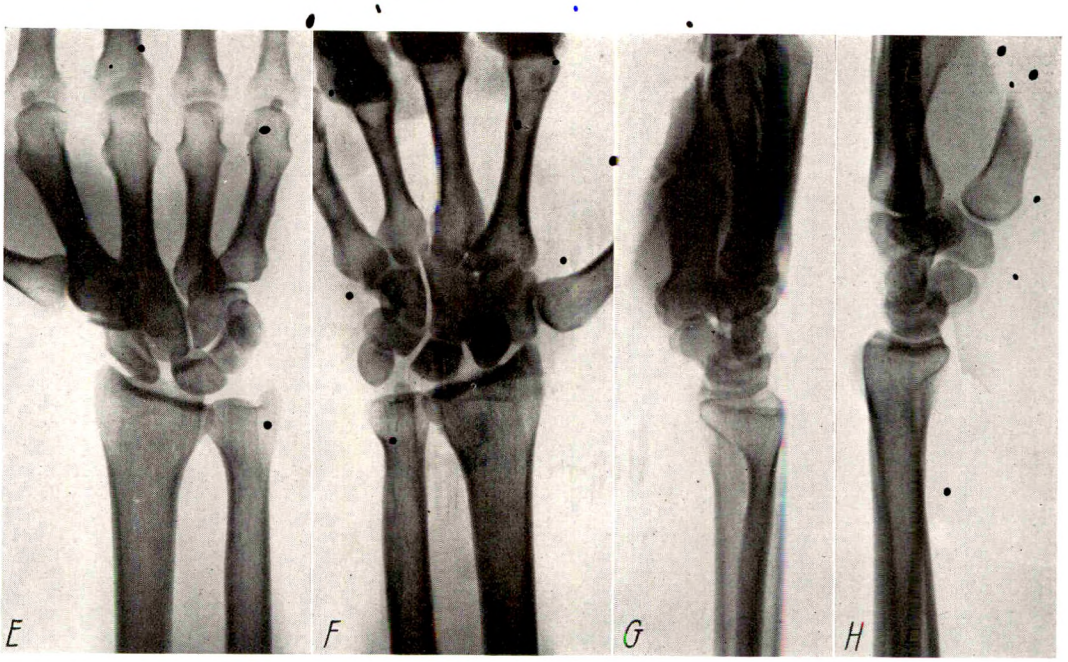


FIG. 3. *E*, posteroanterior view in dorsiflexion; *F*, anteroposterior view in palmar flexion; *G*, lateral view—radio-ulnar; *H*, lateral view—ulnar-radial.

flexed wrist. Fracture of the ulnar styloid occurs not uncommonly in association with Colles fracture and seldom as an entity in itself.

Oblique traumata to the wrist and acute rotational traumata are uncommon. Occupational rotational traumata may cause chronic disability of the wrist.

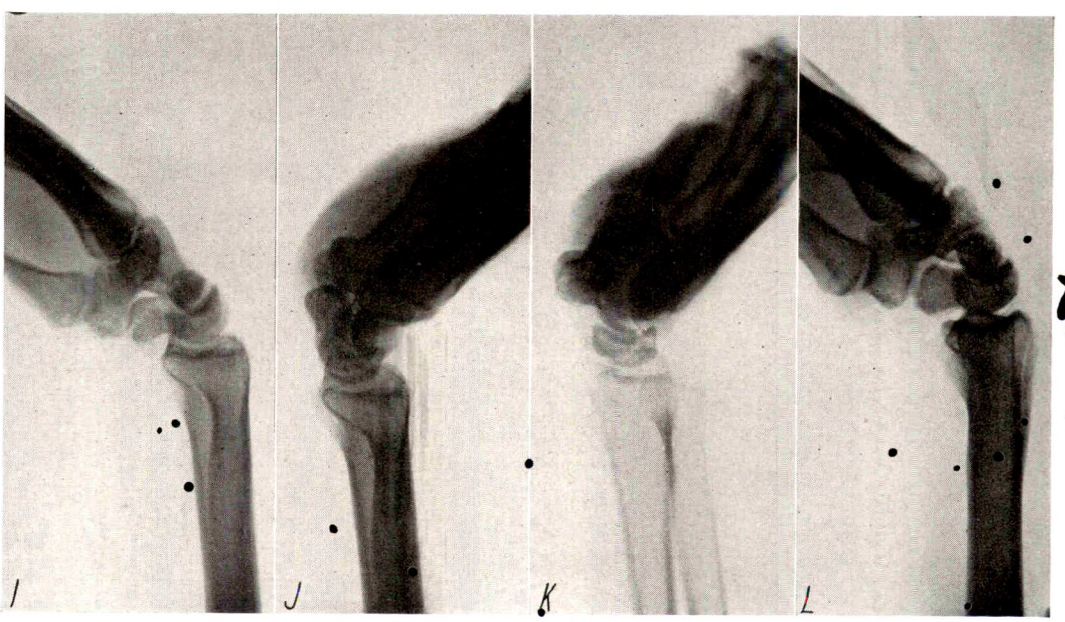


FIG. 3. *I*, lateral view in palmar flexion; *J*, lateral view in dorsiflexion; *K*, lateral view in dorsiflexion and radial abduction; *L*, lateral view in volar flexion and ulnar adduction.

II. THE ROENTGENOGRAPHIC INTERPRETATION OF THE NORMAL WRIST

Interpretation of the normal wrist is ordinarily difficult because of the irregularity and overlapping of the eight carpal bones. The postero-anterior view is usually easily read but the oblique and lateral views offer difficulty (Fig. 3).

The study of the wrist in palmar flexion and dorsiflexion informs us about carpal bone rotations. *In dorsiflexion of the wrist* (to which is always added some radial abduction by reason of obliquity of the scaphoid) the scaphoid is elongated in the lateral view so that it is placed in the line of the long axis of the forearm. The proximal end of the bone shifts volarly and the distal end dorsally, increasing its obliquity. The semilunar rotates dorsally. The greater multangular is distorted. The *posteroanterior view in dorsiflexion* shows the scaphoid to be more in the line of the axis of the forearm. The joint space between scaphoid and semilunar is increased, and the head of the os magnum seems to approach the articular surface of the radius. The metacarpal bases overlie the distal carpal bones.

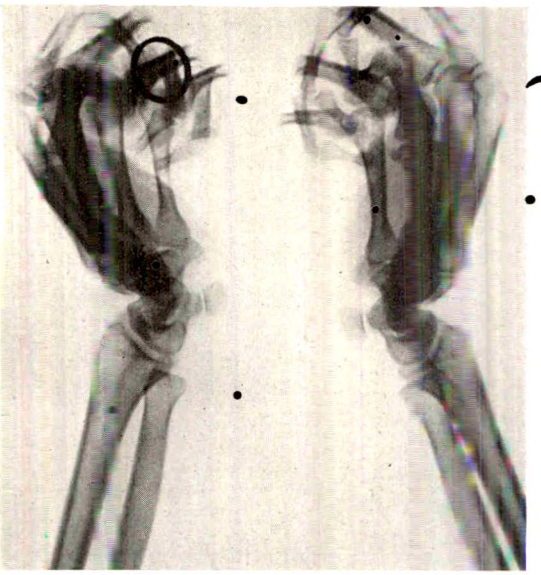


FIG. 3*M*. Oblique ulnar-radial views of the wrist joint of another person. This view shows the pisiform and its articulation with the cuneiform very clearly.

Palmar flexion is carried out in some degree of ulnar adduction because of obliquity of the scaphoid. In the *lateral view in palmar flexion*, it is seen that the scaphoid

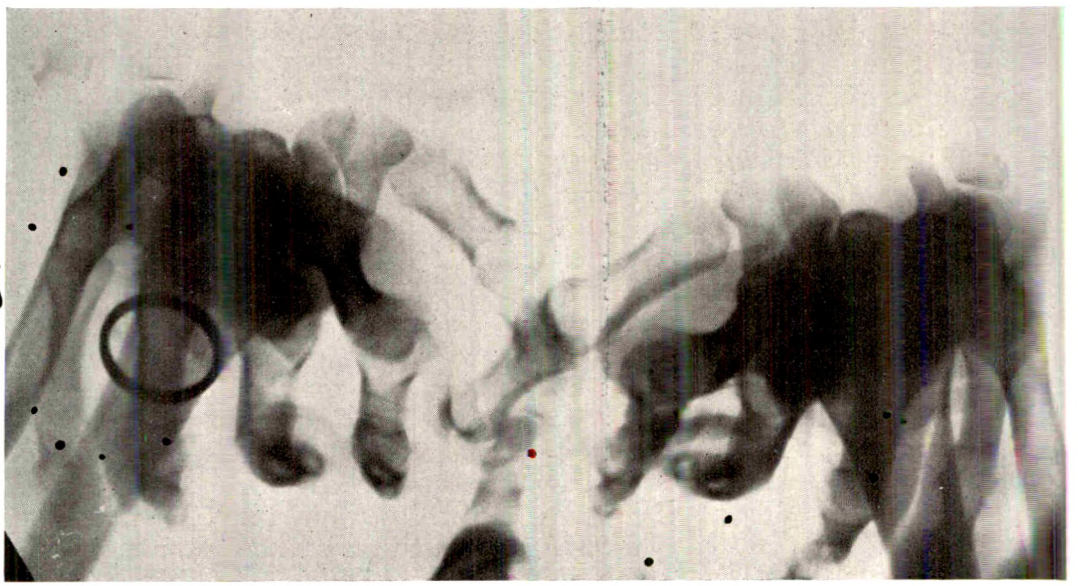


FIG. 3*N*. Carpal canal views of the same patient as in Figure 3*M*. This shows the pisiform, the hook of the hamate, the tuberosity of the scaphoid, the greater multangular, particularly its ridge, and the bony floor of the canal.

is rotated anteriorly, so that it lies dorsovolarly. The semilunar rotates anteriorly at the same time. The greater multangular is well visualized and the articular line between it and the base of the first metacarpal is a little oblique to the longitudinal axis of the forearm. The *anteroposterior view in palmar flexion* shows enlargement with distortion of the greater multangular and other distal carpal bones. The articular line of the greater multangular is placed radially in line with the forearm. The scaphoid is foreshortened, its body overlapped by the tuberosity.

Dorsovolar movement of the wrist occurs about an axis which is slightly oblique. To simplify matters, the wrist, as any other joint, may be thought to have three principal axes: a *transverse* or *radio-ulnar axis*, a *vertical* or *dorsovolar axis*, and a *longitudinal axis*, that which is parallel to the long axis of the forearm. Only the first two of these axes can be important, since there is little motion about the longitudinal axis. Dorsovolar motion of the wrist takes place about a transverse axis, while side to side movement is about a vertical axis. In dorsovolar motion, movement takes place predominantly about the transverse axis to which movement about the vertical axis is added by reason of scaphoid obliquity.

It must be understood that each carpal bone *behaves not as an individual in these various movements of the wrist, but moves in integrated manner with the other carpal bones.* This is quite necessary for normal wrist function. This rotation or change in position of each carpal bone, integrated as it is, is a *rotation in parallel* or *parallel rotation.* This is illustrated by the parallel movements of the scaphoid and semilunar bones in palmar flexion and dorsiflexion. As the wrist flexes palmarly, the scaphoid rotates anteriorly about its transverse axis, becoming dorsovolarly placed, while the semilunar rotates anteriorly on a similar axis. In dorsiflexion, both bones rotate in opposite direction. The other carpal bones follow in similar parallel movement.

Normally there is and can be no dissociation of physiological parallel rotation. The limits of physiological parallel rotation are the limits of motion of the wrist. If such parallel rotation exceeds in degree the *normal* range of rotation for a given position of the wrist, then a *pathological parallel rotation* must exist.

III. ROTATION OF THE CARPAL BONES

It is necessary to study the movements of the carpal bones in the normal wrist and to create experimentally abnormal rotations in the carpal skeleton in order to know and understand clinical abnormal rotations of the carpal bones. The situation is simplified by the fact that it is the rotational movement of only three carpal bones which matters—that of the scaphoid, the semilunar, and the greater multangular. The *scaphoid-semilunar* unit is the more important.

It is easier to imagine rotational movement of a carpal bone if the axes of motion are made to apply to a particular bone and not to the wrist as a whole. These axes are similar to those of the wrist except that the longitudinal axes of the scaphoid and of the greater multangular are oblique to the longitudinal axis of the wrist.

Abnormal rotation of a carpal bone may or may not exceed its limit of physiological rotation. It is more likely that pathological rotation of one or more carpal bones will be seen rather than whole carpal movements as in the normal wrist.

A. Experimental Rotation of the Carpal Bones (Fig. 4 and 5).

1. Rotation of the Scaphoid.

When the scaphoid rotates about its *transverse* axis, the bone moves dorsally and volarly, the direction of rotation being reckoned by the position of the tuberosity. In the posteroanterior view, the tuberosity overlaps the body, with change in the articular relationship between scaphoid and radius. In the lateral radio-ulnar view, the obliquity of the scaphoid is lost. The bone is placed more dorsovolarly, and *is not foreshortened.* The tuberosity is anterior in the more probable anterior rotation of the scaphoid. The intermediate dorsally ro-

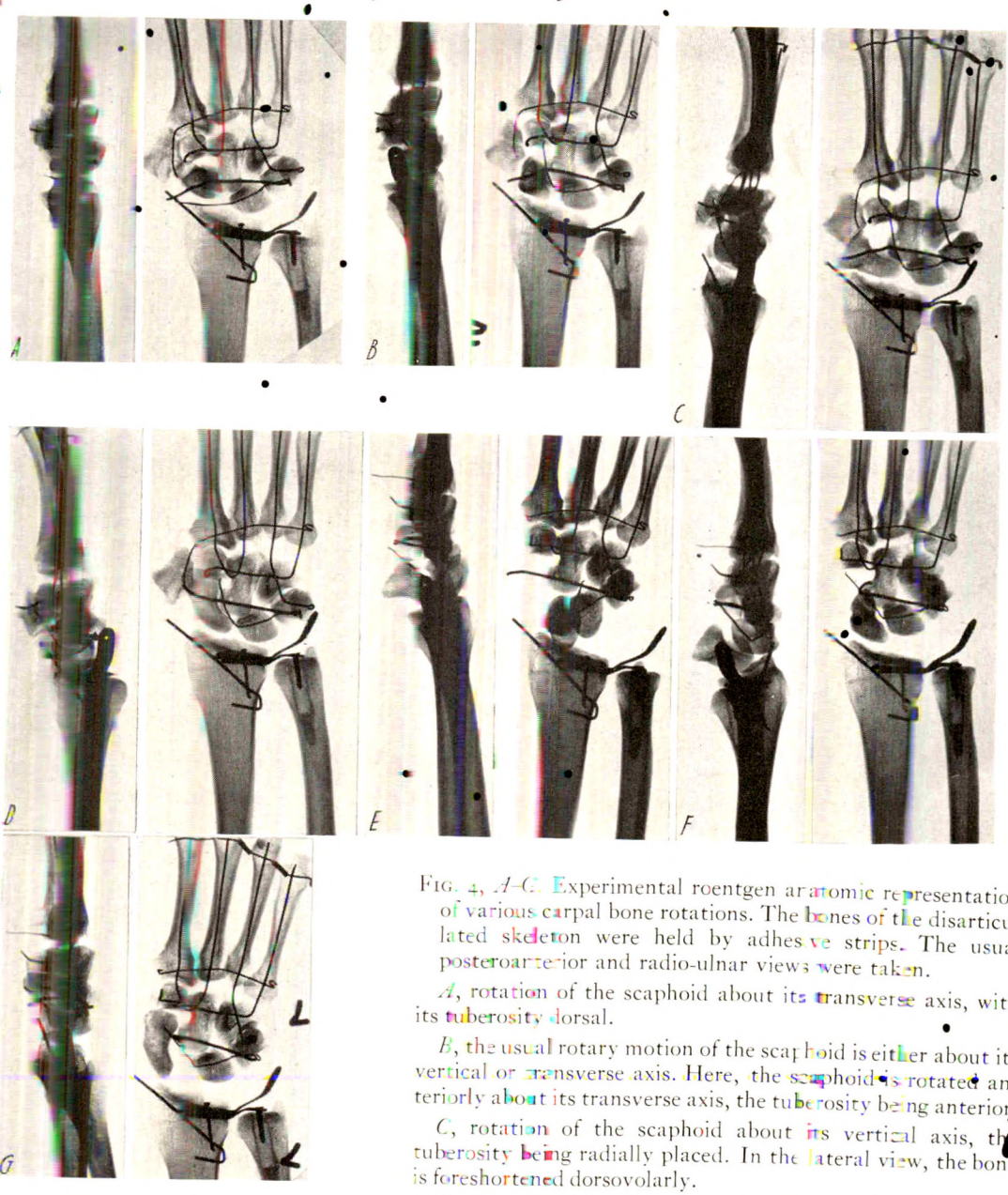


FIG. 4, *A–G*. Experimental roentgen anatomic representation of various carpal bone rotations. The bones of the disarticulated skeleton were held by adhesive strips. The usual posteroanterior and radio-ulnar views were taken.

A, rotation of the scaphoid about its transverse axis, with its tuberosity dorsal.

B, the usual rotary motion of the scaphoid is either about its vertical or transverse axis. Here, the scaphoid is rotated anteriorly about its transverse axis, the tuberosity being anterior.

C, rotation of the scaphoid about its vertical axis, the tuberosity being radially placed. In the lateral view, the bone is foreshortened dorsovolarly.

D, ulnar rotation of the scaphoid on its vertical axis.

E parallel rotation of scaphoid and semilunar bones, the tuberosity of the scaphoid being directed anteriorly by rotation of the bone on its transverse axis. The semilunar bone rotates so that its radial articulating surface faces more posteriorly and its concavity anteriorly.

F anterior dorsovolar gear rotation between the semilunar and scaphoid bones, a movement which is the reverse of the parallel movement shown above. The semilunar bone rotates in a dorsovolar direction, so that its concave surface faces more posteriorly. Rotation of both bones is again about a transverse axis.

G proximal radio-ulnar gear movement of the scaphoid and semilunar bones. Rotation of each bone is about a vertical axis, and the semilunar, sometimes narrowed in lateral view, is rotated ulnarly in the posteroanterior view.

tated scaphoid is in a line with the forearm, and its roentgenographic appearance is much like that of the scaphoid ulnarly rotated about its vertical axis. Rotation of the scaphoid about its *vertical axis* carries it ulnarly or radially. In the posteroanterior view, the scaphoid is placed in the line of the forearm when it rotates ulnarly, and it approaches the radial styloid in radial rotation, lying radio-ulnarly. In the lateral view, the obliquity of the bone is increased on ulnar rotation, so that it lies in a line with the forearm, while it is dorsovolarly placed but *foreshortened* in radial rotation. In radial rotation, there is also widening of the joint space between the scaphoid and greater multangular.

It is more difficult to determine rotation about the *longitudinal axis* of the scaphoid, but it is certain that obliquity of the bone is not changed. Larger degrees of such rotation show change in articular relationship between radius and scaphoid.

Complex rotation is more probable than simple rotation, rotation about the transverse and vertical axes, or all axes being combined. Rotation may, however, be predominantly about one axis.

A dorsovolar position of the scaphoid is noted in the lateral view both in anterior rotation about the transverse axis and in radial rotation about the vertical axis. In rotation about the transverse axis, the bone is seen in full length, while in radial rotation about the vertical axis, the tuberosity of the scaphoid points radially, and hence, the bone seems foreshortened.

2. Rotation of the Semilunar Bone.

The direction of rotation of the semilunar bone is reckoned by the position of its concavity.

The semilunar bone rotates radially and ulnarly on its *vertical* axis.* More of the scaphoid facet is seen on radial rotation in the posteroanterior view, while in the lateral view the semilunar bone becomes narrowed. It rotates dorsally and volarly on its *transverse* axis, so that a varying degree of its radial articular surface is seen in the posteroanterior view. It is possible to determine posterior rotation of the semilunar in the posteroanterior view, whereas the determination of anterior rotation in this view is a little more difficult. In anterior rotation exceeding 90°, the body of the bone overlaps the concavity, so that the latter is poorly represented. Posterior rotation seldom exceeds 90° so that the concavity may be dissociated from the radial articular surface. The lateral view shows this rotation well. Rotation, with anterior dislocation of this bone, occurs predominantly about the transverse axis, to which may secondarily be added rotation about other axes. The semilunar rotates radially and ulnarly in a vertical plane on its *longitudinal* axis. When it rotates radially, the scaphoid facet faces more proximally.

3. Rotation of the Greater Multangular.

The greater multangular is obliquely placed, its long axis running from superior and radial to inferior and ulnar. When it rotates about its transverse axis, it moves dorsovolarly, its obliquity being lost. On its vertical axis, it rotates radially and ulnarly, again with loss of its obliquity. It preserves this obliquity when it rotates on its own long axis.

Change of position of its articulating face with the first metacarpal bone helps to indicate direction of rotation.

To all carpal bone rotations, a variable *translation* may be added, a movement of the rotated bone more likely with severe trauma.

B. Abnormal Parallel and Gear Motions of the Carpal Bones. Experimental and Theoretical Considerations.

The definition of parallel rotation indicates that abnormal parallel rotation must follow the pathway of physiological parallel rotation. Such parallel rotation is abnormal when it is found in a position of the wrist

* Rotation of the scaphoid and semilunar about their vertical axes should duplicate more or less the position of each bone in normal radial abduction or ulnar adduction. There is seeming difference in the roentgenographic appearance of these bones in normal lateral movement and in pathological rotation, but this is due to their greater movement in the latter rotation either experimentally or clinically seen.

The same conception holds true for the position of these bones after rotation about their other axes.

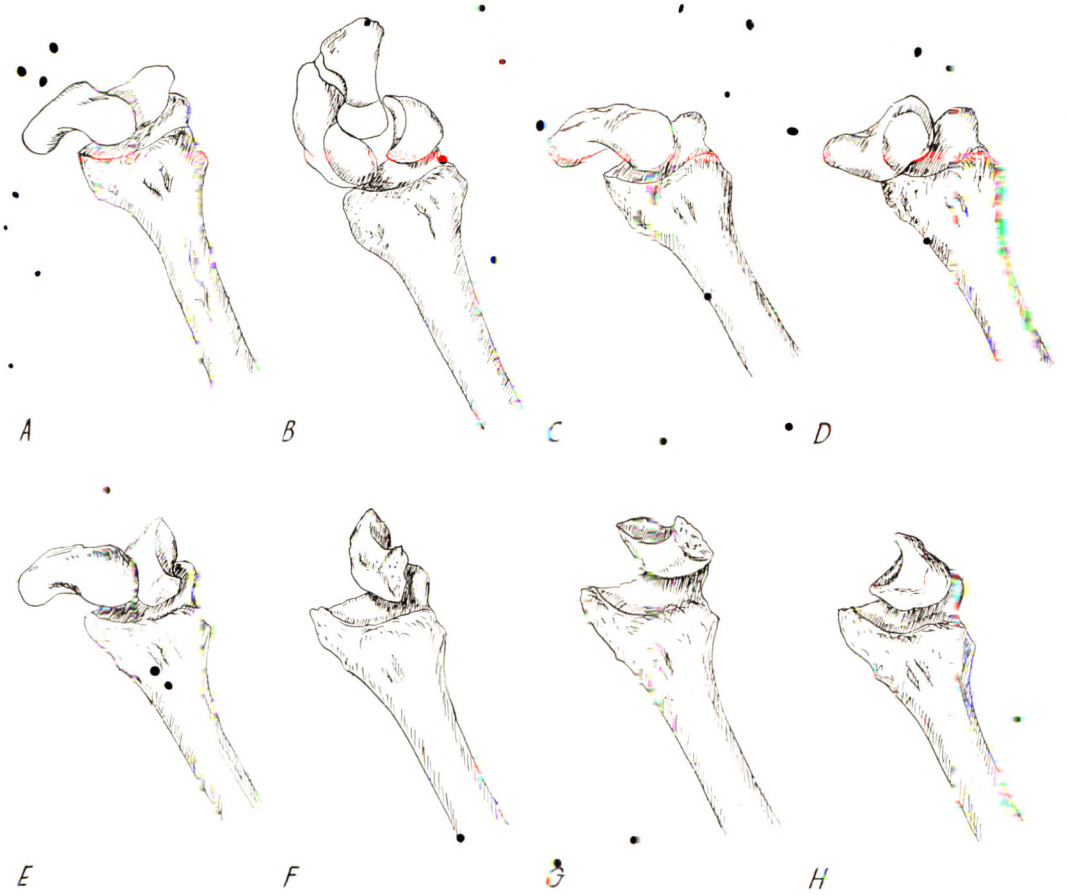


Fig. 5, *A–H*. Three dimensional representations of carpal bone rotations. In each drawing, the eye looks down upon the dorsal surface of the radius.

A, parallel rotation of the scaphoid and semilunar bones. The tuberosity of the scaphoid is directed anteriorly, while the convexity of the semilunar is dorsally or posteriorly placed.

B, proximal radio-ulnar gear movement of the scaphoid and semilunar.

C, anterior rotation of the scaphoid about its transverse axis.

D, radial rotation of the scaphoid about its vertical axis.

E, anterior dorsovolar gear movement between the scaphoid and semilunar.

F, posterior rotation of the semilunar bone about its transverse axis. The concavity of the semilunar bone faces posteriorly and the convexity anteriorly.

G, ulnar rotation of the semilunar bone about its vertical axis.

H, anterior rotation of the semilunar bone about its transverse axis.

where it normally does not occur. Since abnormal parallel rotation follows a physiological pathway, it will take a lesser or minimal force to create it than it will to dissociate parallel motion *in reverse parallel or gear movement*. The loosely jointed wrist may show such abnormal parallel rotation more easily than a firmly jointed wrist.

In abnormal parallel rotation of the scaphoid and semilunar, the two bones rotate about similar axes in similar direction, while in gear movement these two bones (or any other two bones entering into such movement) rotate in opposite direction on similar axes.

Gear rotation may take place anywhere in the carpus, but is more likely between two carpal bones which usually have a fair range of motion.

There is one site in the carpus at which gear rotation of one bone on another is probable—*gear rotation between the scaphoid*

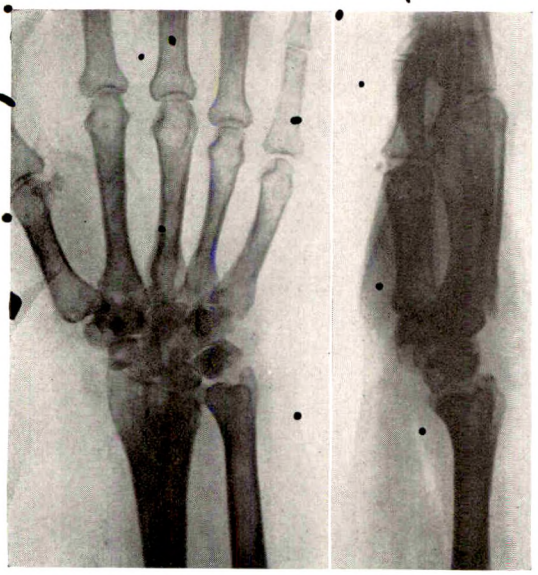


FIG. 6. A roentgenogram of the arthritic wrist of a female, aged fifty-five, illustrating parallel rotation between the semilunar and scaphoid bones, the force creating this rotation being the gradual advance of the head of the os magnum. The scaphoid is rotated radially about its vertical axis, and also on its longitudinal axis, while the semilunar is rotated ulnarly about its vertical axis. The tuberosity of the scaphoid is made to approach the radial styloid by this rotation. The lateral view shows a foreshortened scaphoid placed dorsovolarly. The semilunar is not rotated about its transverse axis.

and the semilunar. Rarer gear rotations may be possible between the scaphoid and greater multangular bones and between the hamate and cuneiform bones.

The term, gear rotation, is proper, since the movement of the bones on each other is as a movement of gears.

In experimental parallel and gear motions of the scaphoid and greater multangular, the standard is the rotation of each bone about a transverse axis in normal palmar and dorsiflexion of the wrist. The combinations in parallel rotation are four. When both bones are rotated anteriorly about their transverse axes, the roentgenographic appearance is like that of the normal wrist in palmar flexion. The greater multangular is elongate, pear shaped and distorted in the posteroanterior view and the articulating face with the first metacarpal is radial. The scaphoid is overlapped by its tuberosity. In the lateral view, the parallelogram of the greater multangular is tilted more volarly, following the anteriorly placed tuberosity of the scaphoid. Posterior rotation about the transverse axis of each bone simulates the position of each bone in dorsiflexion of the wrist. Parallel rotation about the vertical axis is also possible.

The possible gear movements in the anatomical specimen are the gear movement in which the scaphoid is anteriorly rotated about its transverse axis, while the greater multangular rotates posteriorly about a similar axis, and its opposite; and the gear movement in which the scaphoid rotates ulnarly about its vertical axis, while the greater multangular rotates radially on a similar axis, and the opposite of this. The most possible of these gear movements is the one in which the greater multangular rotates volarly, and the scaphoid dorsally. Since dorsal rotation of the scaphoid is uncommon, this gear rotation will be a most unusual one.

Four *parallel rotations of the scaphoid-semilunar unit* are possible. These are parallel anterior or posterior rotation of both bones on a transverse axis and parallel radial or ulnar rotation on a vertical axis. It is quite possible that such parallel rotation may take place on two axes rather than one (Fig. 6).

Four possible *gear movements* may be made to take place *between the scaphoid and semilunar bones*. Two are in a dorsovolar direction (*anterior dorsovolar* and *distal or posterior dorsovolar gear movement*), the movement of the tuberosity of the scaphoid predicating the terms anterior or posterior. Both bones rotate about their transverse axis in a reverse parallel movement, and therefore, a gear movement. In the anterior dorsovolar gear movement, the tuberosity of the scaphoid is directed anteriorly and the concavity of the semilunar faces posteriorly.

The other two gear motions are in a radial and ulnar direction, the probable one being a *proximal radio-ulnar gear* motion,

the improbable being a *distal radio-ulnar gear* motion. The terms *proximal* and *distal* refer to the line of force. It has been pointed out that a distally directed force seems unlikely. In the *proximal radio-ulnar gear movement*, the two bones diverge, rotating about their vertical axes. The semilunar bone is the unstable element of this gear movement, tending to rotate anteriorly as a secondary thing. Spread of divergent force may fracture both styloid processes with consequent dislocation of the wrist.

These proposed terms are satisfactory because they indicate direction in which rotation takes place by indicated force. The word *distal* in *distal or posterior dorsovolar* gear motion is meant to indicate the position of the tuberosity, the distal position being more likely than the posterior.

Gear movement may be complex rather than simple and proximal radio-ulnar gear movement may be combined with anterior dorsovolar gear movement.

Consider the gear movement again as a reverse parallel movement. The proximal radio-volar gear movement, as well as the distal or posterior dorsovolar gear movement, seems derived from the parallel movement of the scaphoid and semilunar in the position of dorsiflexion. The anterior dorsovolar gear movement seems the derivative of the parallel movement of both bones in the position of palmar flexion. Each gear position is, however, created by a force which puts the wrist in a greater or lesser position of dorsiflexion. Whether the anterior dorsovolar gear is produced in a position of lesser dorsiflexion is problematic, since the angle of such dorsiflexion cannot be measured at the time of injury.

It seems certain that the proximal radio-ulnar gear movement is less stable than the anterior or posterior dorsovolar gear movement. The proximal radio-ulnar gear is difficult to differentiate from the posterior or distal dorsovolar gear movement, since the position of the scaphoid is about the same in each. The position of the scaphoid is therefore not the determining factor in differentiating between these two gear motions. Rather, it is the position of the semilunar. The ulnarly rotated semilunar of the proximal gear motion may easily dislocate anteriorly, converting this gear into the distal dorsovolar gear. It is more difficult to interpret the position of the semilunar in the posteroanterior roentgenogram, and therefore it is less certain which type of gear motion is present.

It is important to appreciate that these gear motions are not haphazardly created, but arise from physiological positions or pathways of motions of the participating bones.

It is the suddenly advancing head of the os magnum in a wrist subjected to *relatively maximal trauma* which creates gear motions between the semilunar and scaphoid bones.

Several things may happen, the resultant disability depending on the manner of concentration of force at a particular part of the articulation the scaphoid and semilunar make with the caput of the os magnum. If the articular area of the scaphoid bears the brunt of the proximally flowing force, a mid-carpal dislocation may take place, the scaphoid fracturing at its waist. The proximal part of the scaphoid goes with the usually anteriorly dislocated semilunar bone, while the distal part of the scaphoid rotates ulnarly as a gear against the semilunar and the attached proximal part of the scaphoid. *Reverse mid-carpal dislocation* is also possible, the semilunar and the attached proximal half of the scaphoid rotating dorsally. Wedge action of the caput of the os magnum may also give translation of the parts. The head of the os magnum becomes placed nearer the radius, so that there is shortening of the carpus. The scaphoid may break without particular displacement and the semilunar rotate alone, or the broken distal half of the scaphoid may rotate ulnarly without accompanying semilunar rotation (Fig. 7).

If the brunt of the blow is borne by the scaphoid-semilunar joint, gear motion, simple or complex, is produced. If the semilunar articulating area bears the greater force of blow, this bone either rotates or dis-

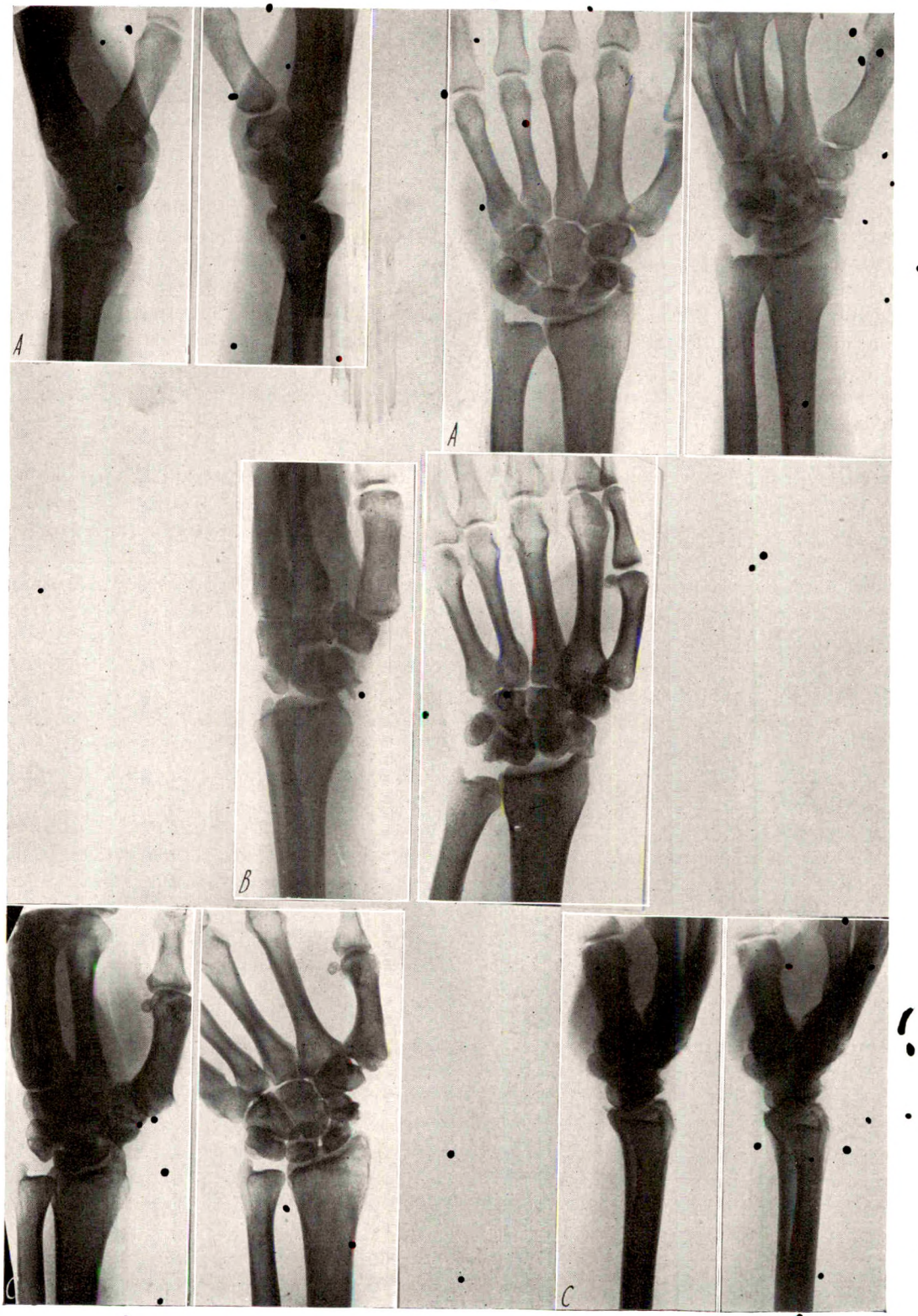


FIG. 7A–7C (See opposite page for description.)

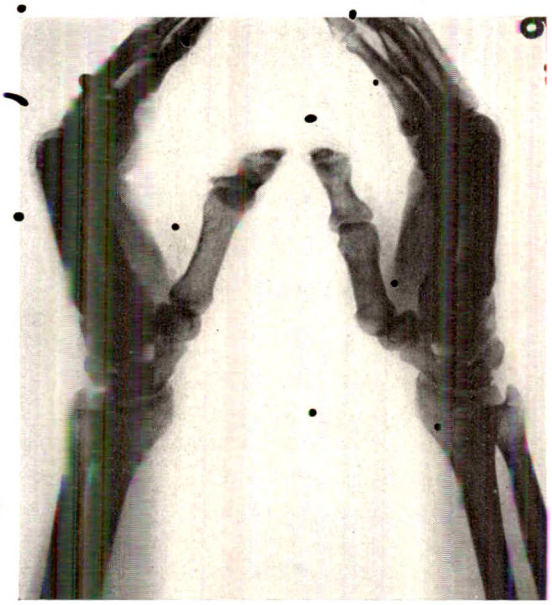


FIG. 8. The loose right wrist of a female, aged thirty-one, had been painful for a week without trauma. Slight clicking sounds were heard on motion of the wrist as if one bone subluxated on another. This was most evident over the semilunar and scaphoid bones. When the carpal bones were squeezed together, release of pressure caused separation of the carpal bones with clicking. Radial abduction was increased so that the base of the first metacarpal impinged upon the radial styloid. This motion was painful and the scaphoid, which showed increased rocking motion when it was grasped, clicked on radial abduction. A difference in obliquity of each scaphoid is seen. The symptomatic right wrist shows a lesser obliquity of the bone.

painful and accompanied by clicking. Even the hamate, which is usually firmly bound, may be lax. The bones are easily squeezed together, with resultant clicking on release of compression.

The carpal bones may sometimes show vacuolization.

It is possible that the scaphoid of the loosely jointed wrist may be rotated gradually by a *kinesiological imbalance between the thenar and hypothenar muscles*, in favor of the former. A case of asymptomatic rotation of the scaphoid was seen in a hand whose weakness was relieved by scalenotomy. The opposite wrist showed similar rotation. There seemed to be no causal connection between the spasm of the scalenus anticus muscle and the rotation of the scaphoid. Yet, when a similar rotation of the scaphoid was seen in another woman in whom scalenotomy was done, it appeared that, while coincidence could not be ruled out, lacking a larger series, cause and effect association might be considered. In the scalenus anticus syndrome, it is more usual that the ulnar nerve is involved than the median or radial nerves. This gives the muscles of the hand innervated by the median nerve a *relative* or *actual* kinesiological predominance over the muscles innervated by the ulnar nerve. The word "*relative*" is stressed, for, while the scalenus anticus syndrome may be clinically unilateral, it may yet be occultly bilateral. This theory must include the muscle imbalance caused by any motor lesion of the ulnar nerve, and is not meant to be restricted to the scalenus anticus syndrome alone.

The wrist must be looked at a little differently, so that the bony structures of the carpal canal are accented. The transverse carpal ligament is attached in good part to the tuberosity of the scaphoid, the ridge

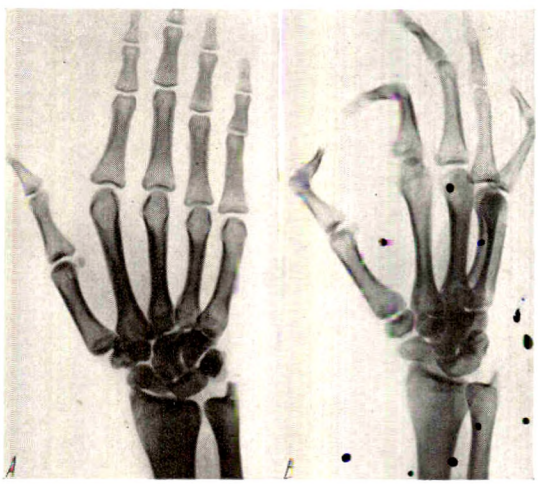


FIG. 9, *A* and *B*. Anteroposterior and oblique roentgenograms of the right wrist showing asymptomatic radial rotation of the scaphoid with parallel rotation of the semilunar bone about the transverse and vertical axes in a girl, aged seventeen, with a scalenus anticus syndrome relieved by scalenotomy. The major disability was weakness of the ulnar interosseus muscles.

locates anteriorly with or without movement of the scaphoid. The semilunar may of course undergo further and more complex rotation after it has been dislocated anteriorly.

The cuneiform bone in proximal radio-ulnar gear movement and in the mid-carpal dislocation may be forced more ulnarly (posteroanterior view) by spread of the force which creates these disabilities of the wrist. The lateral view may show dorsal rotary luxation of this bone.

C. Clinical Rotation of the Carpal Bones.

1. Rotation of the Scaphoid and the Loosely Jointed Wrist.

In the loosely jointed wrist, rotation of the scaphoid and associated parallel rotation of the semilunar and sometimes the greater multangular bone may be caused by several factors other than axial force. It is easier for a loosely held scaphoid to rotate than a firmly bound one, whether the trauma is axial, occupational, torsional, or otherwise. This rotation need not be fixed. It may not be demonstrated roentgenographically, although the bone can be easily rotated anteriorly when it is grasped between its body and tuberosity. This is a *dynamic* rotation of the scaphoid, since such rotation is created by active or passive movement.

We have examined 7 patients, 4 of them women, with loose or lax wrist. This condition must be quite common, and is asymptomatic as a rule. We stress laxity of the scaphoid, since it is the most important carpal bone clinically (Fig. 8).

The wrist easily becomes painful when subjected to any strain, occupational or otherwise; or the trauma may be the usual fall on the ball of the hand. The rotation may be recognized clinically by anterior prominence of the tender tuberosity of the scaphoid, by a more than usual passive foreshortening of the first metacarpal bone, and by increased hollowing of the "snuff box." The joint between the greater multangular and the first metacarpal bone may be lax.

In the loosely jointed wrist, one carpal bone may be subluxated slightly on its adjoining bone by a passive dorsovolar movement of the bone. This movement may be

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Fig. 7*A*. These roentgenograms show an old, ununited fracture of the distal half of the scaphoid. The slightly oblique view dissociated the fractured scaphoid from the radial styloid, and proved that the latter was not fractured, a probable finding in the posteroanterior view. The tuberosity was displaced proximally and ulnarly, and, clinically, filled the anatomical snuff box. There was little resultant clinical disability from this fracture. This is a forerunner of the more typical mid-carpal dislocation.

Fig. 7*B*. Mid-carpal dislocation by a wedge action of the advancing head of the os magnum. The anteriorly rotated semilunar bone is accompanied by the proximal half of the scaphoid, while the distal part of the scaphoid, rotated ulnarly and proximally, becomes placed nearer the radial styloid. While the movement of the semilunar and proximal half of the scaphoid is parallel, the movement of the two as a unit is as a gear against the distal half of the scaphoid. This mid-carpal dislocation is an analogue of proximal radio-ulnar gear motion. The original rotation is about a vertical axis, as shown by the position of the distal part of the scaphoid. The position of the semilunar and of the attached proximal half of the scaphoid is unstable, and continuation of force places both in the more stable anterior position by a secondary rotation about a transverse axis, the cuneiform following. This fracture dislocation is the usual thing, while pure gear movement between semilunar and scaphoid by acute trauma is unusual. The unit of scaphoid and semilunar breaks at its weakest point—the middle of the scaphoid. The cuneiform bone whose dorsal aspect shows a chip fracture, also shows rotary dorsal luxation about its transverse axis. The caput of the os magnum shows a relative dorsal dislocation. Roentgenographic examination of the left elbow showed a fracture of the lateral and anterior aspect of the radial head by more proximal continuation of trauma.

Fig. 7*C*. This is an instance of reverse mid-carpal dislocation. The tuberosity of the fractured scaphoid is rotated in the same manner shown in Figures 7*A* and 7*B*. However, the proximal half of the scaphoid and the semilunar are rotated posteriorly or dorsally about a transverse axis, to a lesser degree than anterior dislocation in the usual mid-carpal dislocation. The lateral views were taken about two months after the posteroanterior view and the lateral oblique views. The semilunar bone is narrowed in its height, indicating persisting rotation of this bone about its vertical axis.

of the greater multangular, and hook or hamulus of the hamate. Arising from this ligament are the obliquely directed thenar muscles, and the hypothenar muscles. Only a part of the abductor pollicis brevis arises from the tuberosity of the scaphoid, so that it is only through the intermediacy of the transverse carpal ligament that the thenar muscles can pull on this tuberosity. The hypothenar muscles arise from the ligament and from the pisiform and hook of the hamate. The flexor carpi ulnaris, a muscle innervated by the ulnar nerve, ends in part on pisiform and hamate.

Oblique pull of the stronger thenar muscles on the tuberosity of the scaphoid can rotate the scaphoid anteriorly and radially (in women more probably than in men, and in those women whose wrists are lax enough) although the mechanical advantage is poor; yet it is greater for the thenar muscles than it is for the hypothenar muscles. The pull of the latter on the hook of the hamate can rotate the hamulus distally. Whether this is so or not can be determined in hands with median ulnar nerve predominance. The use of the special carpal canal view described by Hart may help.

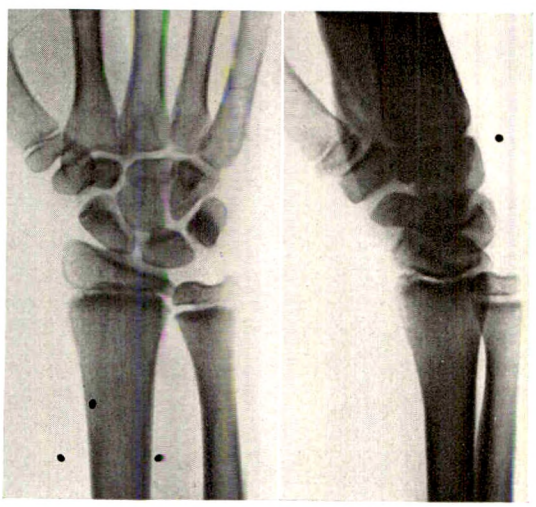


Fig. 10. A boy, aged seventeen, with unilateral symptomatic rotation of the scaphoid after injury. The mechanism of injury was most probably axial. The dorsum of the wrist was swollen with tenderness over scaphoid and semilunar bones. Proximally directed pressure in line of first metacarpal gave pain in the slightly hollowed snuff box. The lateral oblique roentgenogram of the right wrist shows rotation of the scaphoid about its transverse axis, with parallel rotation of the semilunar bone about a similar axis. The joint space between scaphoid and greater multangular is widened. The posteroanterior roentgenogram shows nothing noteworthy. The roentgenograms of the left wrist were normal.

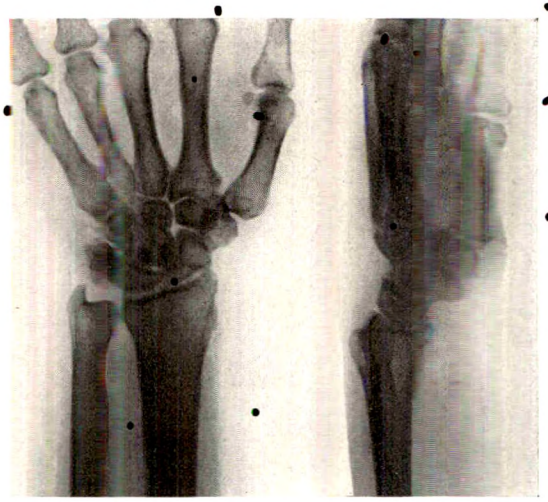


Fig. 11. Parallel rotation of scaphoid and semilunar by axial trauma. Both bones are rotated about their vertical and longitudinal axes with lesser rotation about their transverse axis. The radial styloid is broken. The normal wrist showed potentiality of rotation about the longitudinal axis, for more of the inferior articular surface of the scaphoid could be seen.

This belief in the kinesiologic rotation of the scaphoid needs the amplification and confirmation of more experience (Fig. 9, 10 and 11).

2. Parallel and Gear Rotation of Scaphoid and Greater Multangular.

In a previously published paper,[3] gear rotation between the scaphoid and greater multangular was described. A second case, quite similar to that one was seen. Both cases were examined in the light of the more extensive studies now made.

In the published case, there was anterior rotation of the scaphoid about its transverse axis, together with slight posterior rotation of the semilunar, or, in other words, a mild anterior dorsovolar gear movement between semilunar and scaph-

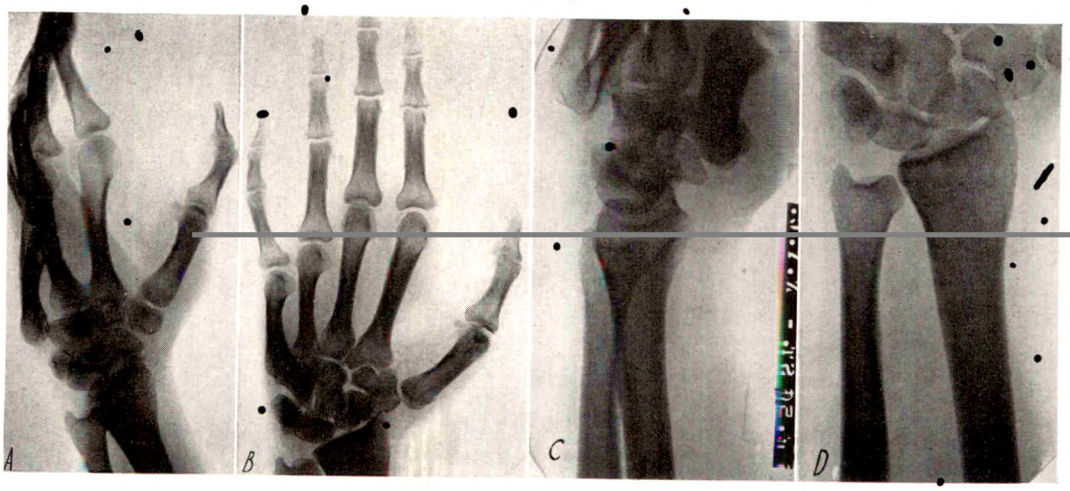


FIG. 12. Anteroposterior, oblique and lateral roentgenograms of the left wrist, illustrating double parallel anterior rotation of the carpus—scaphoid with greater multangular and scaphoid with semilunar. The dorsal dislocation of the bases of metacarpals IV and V is obvious. A distal chip fracture of the os magnum is seen better in the original roentgenograms. The ulnar shift of the carpus is more apparent than real. The wrist is in radial deviation with approximation of the base of the first metacarpal to the radial styloid.

oid, while scaphoid and greater multangular moved in parallel. In the later case, a double parallel rotation is seen, a movment normally present in the palmarly flexed wrist. In both cases, it is questionable if subluxation of the base of the first metacarpal exists. This is more apparent than real because rotation of the scaphoid gives foreshortening of the first metacarpal bone (Fig. 12).

We cannot then say that we have observed gear movement between scaphoid and greater multangular clinically.

3. Rotation of the Semilunar Bone

We have regarded the semilunar bone as the relative fulcrum of the wrist. It follows as a corollary that its rotation or dislocation is relatively uncommon (even though semilunar disability ranks second to scaphoid disability). It should be presumed that, in rotation or dislocation of the semilunar bone, other disability by axial trauma may be found, particularly proximal radio-ulnar gear movement, or anterior dorsovolar gear movement, single or combined. Look carefully at the position of the scaphoid in semilunar rotation or dislocation (Fig. 13).

Our own experience and the illustrations (reproduced in textbooks) of instances of semilunar bone dislocation or of perilunar dislocation, in which the semilunar bone is said to remain in place have confirmed our belief that if there is a rotation or dislocation of the semilunar bone, it is often a part of a gear movement which has gone unrecognized.

It would be well to avoid the use of roentgen-anatomic terms, such as perilunar dislocation, because the expression gives no concept of creating force. Dislocation about the supposedly stable bone may be more apparent than real, especially if a milder degree of rotation goes unrecognized.

The treatment of these complex parallel and gear rotations should be manipulative. It is probable that correction of rotation of one bone may correct rotation of its associated bone. Yet manipulation of a simple anteriorly dislocated semilunar may result in a mild post-manipulation anterior dorsovolar gear movement (Fig. 14).

Rotation of the scaphoid is most probably not unusual after removal of the semilunar. Subluxation of the os magnum on the scaphoid is secondary to the rotation of the scaphoid and follows by a gliding movement. Ordinarily, at least one-half of the articular surface of the os magnum is covered by the scaphoid; as the scaphoid ro-

tates, more of the head is uncovered. Ultimately, there may be spontaneous derotation of the scaphoid (Fig. 15).

4. Rotation of the Hamate. Gear Rotation between Hamate and Cuneiform.

This is an unusual form of gear rotation, the existence of which was not considered until a case was encountered (Fig. 16).

The roentgenograms of the wrist showed an anterior and radial dislocation of the hamate. The articular bases of the fourth and fifth metacarpals were well visualized in the posteroanterior view, with foreshortening of each bone. The cuneiform was rotated ulnarly to a lesser degree and this was well brought out in comparative lateral roentgenograms of the normal left

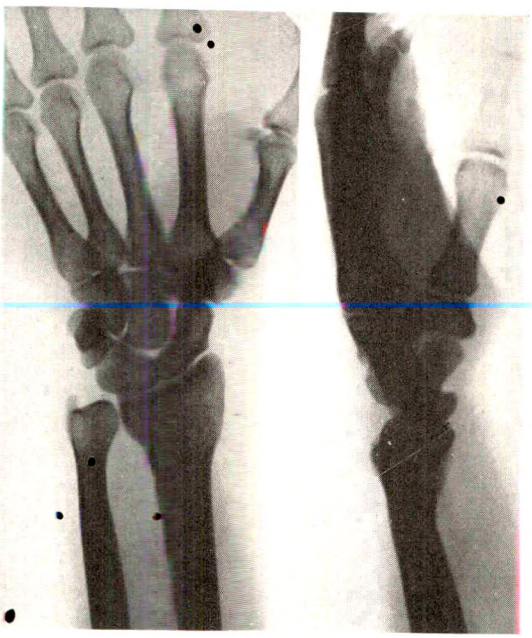


FIG. 13. Posteroanterior roentgenograms show rotation of the scaphoid ulnarly about its vertical axis, so that the bone is placed in the line of the forearm. The lateral view also demonstrated increased obliquity of the scaphoid. The semilunar bone was anteriorly rotated with relative dorsal dislocation of the caput of the os magnum; there was also some rotation about its vertical axis. Either a proximal radio-ulnar gear movement or a distal dorsovolar gear motion, or both, took place. Open reduction of the wrist after failure of manipulation corrected the anterior rotation of the semilunar, the obliquity of the scaphoid being unchanged.

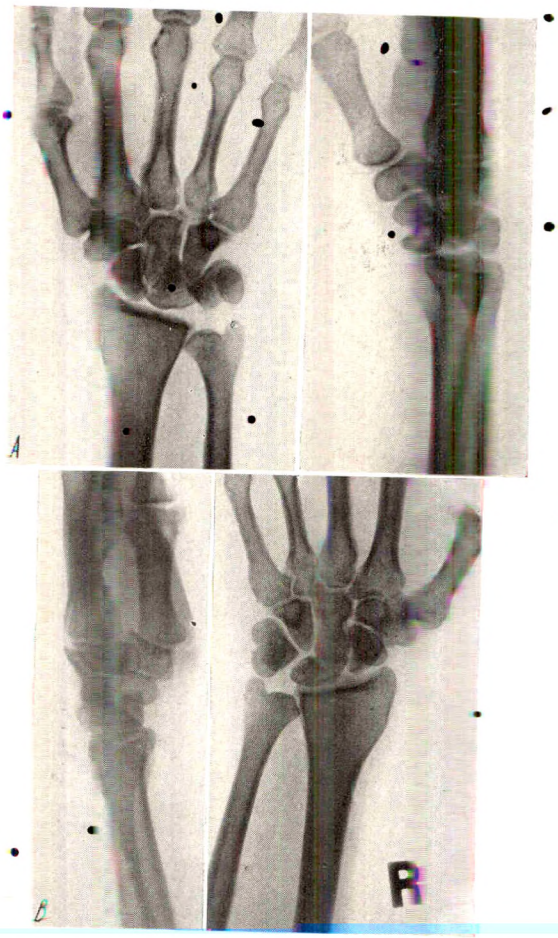


FIG. 14*A*. A painter, aged forty-three, referred by Dr. S. L. Chalfin, fell on both outstretched hands from a stepladder. He dislocated both semilunar bones anteriorly. The right wrist, which was more seriously injured, showed *parallel anterior rotation of semilunar and scaphoid about the transverse axis of each bone*, with anterior dislocation of the semilunar. The caput of the os magnum was relatively dislocated dorsally and was nearer the radial articular surface than it should properly be. Roentgenograms taken on July 20, 1940.)

FIG. 14*B*. Each dislocated semilunar bone was easily reduced by manipulation under general anesthesia. The left semilunar bone was set normally. The right semilunar bone was overcorrected, so that its concavity faces posteriorly, with persisting relative marginal luxation of the caput of the os magnum. The rotation of the scaphoid was unchanged. Therefore, a parallel anterior rotation of the semilunar and scaphoid was converted into an anterior dorsovolar gear movement of these two bones. The right wrist, at the end of October, 1940, still showed a fullness dorsally over the region of the semilunar, and tenderness of the area. Both wrists had about equal motion, 15° of dorsiflexion and 10° of palmar flexion.

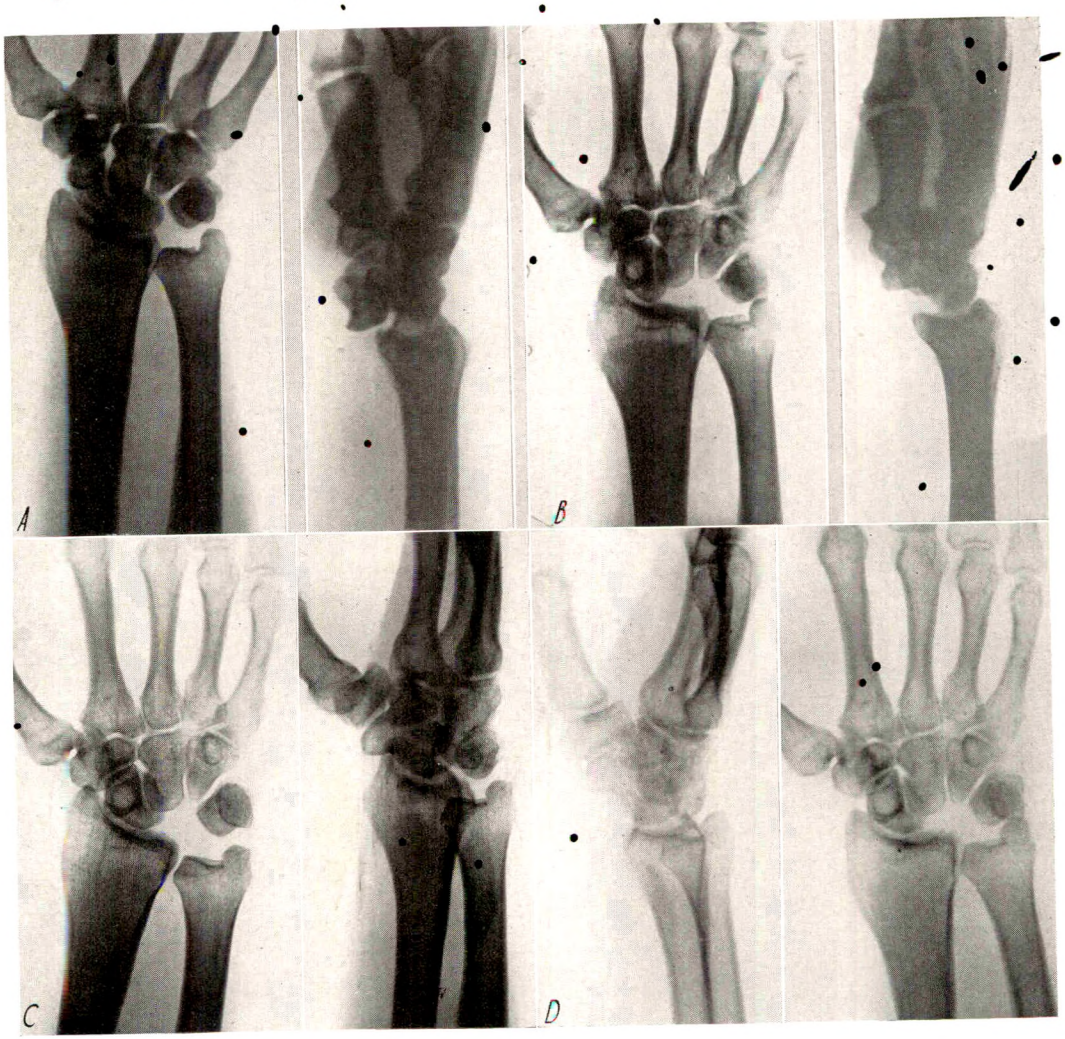


FIG. 15*A*. Posteroanterior and lateral roentgenograms of the right wrist taken on April 21, 1938. In the lateral view, the semilunar bone is dislocated anteriorly and rotated through an arc of 90°. Normal obliquity of the scaphoid is seen. In the posteroanterior view, the semilunar bone is rotated, so that more of its radial (convex) articular surface is seen. (This is the typical appearance of an anteriorly dislocated semilunar in the posteroanterior view.) The position of the scaphoid is normal. There is relative dorsal dislocation of the caput of the os magnum.

FIG. 15*B*. Posteroanterior and lateral roentgenograms of the right wrist taken on August 17, 1938, about four months after the operative removal of the semilunar bone. The scaphoid is rotated, so that the bone is now placed dorsovolarly in the lateral view rather than obliquely. The tuberosity of the scaphoid is more anterior than normal. In the posteroanterior view, the tuberosity overlaps the body of the scaphoid and there is some diminution of the distance between the radial styloid process and the base of the first metacarpal bone. The greater multangular has rotated in parallel with the scaphoid. There is subluxation of the head of the os magnum. Spotty decalcification of the carpus and transverse decalcification of the lower end of ulna and radius are present. Note the sun-ray appearance of the articular lines which seem to radiate from the semilunar space.

FIG. 15*C*. Oblique and posteroanterior roentgenograms of the right wrist taken on July 25, 1939. The findings in the posteroanterior view are similar to those of Figure 15*B* but the distance between the radial styloid and the base of the 1st metacarpal bone is further diminished. The more dorsovolar position of the scaphoid and the anterior prominence of the tuberosity of the scaphoid is well seen in the oblique view. The anterior and parallel rotation of the greater multangular is well demonstrated in this view. The subluxation of the head of the os magnum is again seen. There is now no decalcification.

wrist. This rotation was indicated by increased width of the more proximal part of the joint space between cuneiform and semilunar, by increased obliquity of the articular line between cuneiform and hamate, and by approximation of the cuneiform to the bases of the fourth and fifth metacarpals.

Both bones had rotated about their transverse axes.

It was first thought that a *gliding movement* of cuneiform and hamate had taken place. This movement is possible between cuneiform and hamate, and between os magnum and hamate. Rather, ulnar rotation of the cuneiform took place, the bone using the most distal and ulnar part of the hamate (the ulnar ledge) as a fulcrum.

This is not the first instance of rotation of the hamate encountered.

The hamate is so firmly bound that rotation about its vertical or longitudinal axis need scarcely be considered. The bone rotates radially on its longitudinal axis, direction of rotation being *opposite* to the position of the hamulus. Rotation about its vertical axis does not change the position of the hamulus. The position of the large articular face with the os magnum may indicate direction of this rotation.

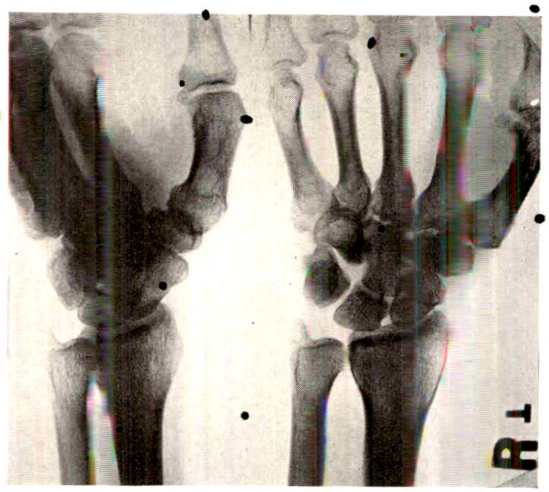


FIG. 16. Gear rotation between the hamate and cuneiform. The hamate is anteriorly and radially dislocated, while the cuneiform is rotated ulnarly. Both bones have rotated about their transverse axes.

IV. CERTAIN INJURIES OF THE RADIAL AXIS

1. It has been stressed that in the axial transmission of force a halt to such force may be made at one of several points. One such point is the fracture of the base of the first metacarpal bone—Bennett's fracture. This fracture (uncommon in childhood) is well known and needs no discussion. Luxation of the base of the first metacarpal may sometimes take place.

Injury of the greater multangular may be isolated or associated with other radial axis fractures, such as fracture of the scaphoid of the base of the first metacarpal bone. In the series of roentgenograms examined, fracture of the greater multangular occurred once as an isolated fracture, once with dislocation of the metacarpophalangeal joint of the thumb, and once with Bennett's fracture. This bespeaks a singular immunity from trauma for the greater multangular, and this is so for three reasons. It is not a superficial and hence vulnerable bone. The trauma is taken up by the base of the first metacarpal bone or more proximally. Lastly, the greater multangular has a strong cortex which protects it against break. The obliquity of the line of force makes associated fracture of the lower end of the radius uncommon.

2. *Injury of the Carpometacarpal Joint the Thumb in Old Persons.* We have observed and treated 2 cases of injury to the carpometacarpal joint of the thumb in old persons (over sixty) in whom an antecedent asymptomatic osteoarthritis became symptomatic by a fall on the radial side of the ball of the hand. This joint is quite prone to such osteoarthritis. In both patients it is believed that an *infraction fracture* of the joint took place, although

FIG. 15*D*. Roentgenograms of May 6, 1940, showing partial spontaneous derotation of the scaphoid with restoration of scaphoid obliquity. The wrist is in a little more dorsiflexion than in Figure 15*C*, which may account for some of the increase in obliquity of the scaphoid.

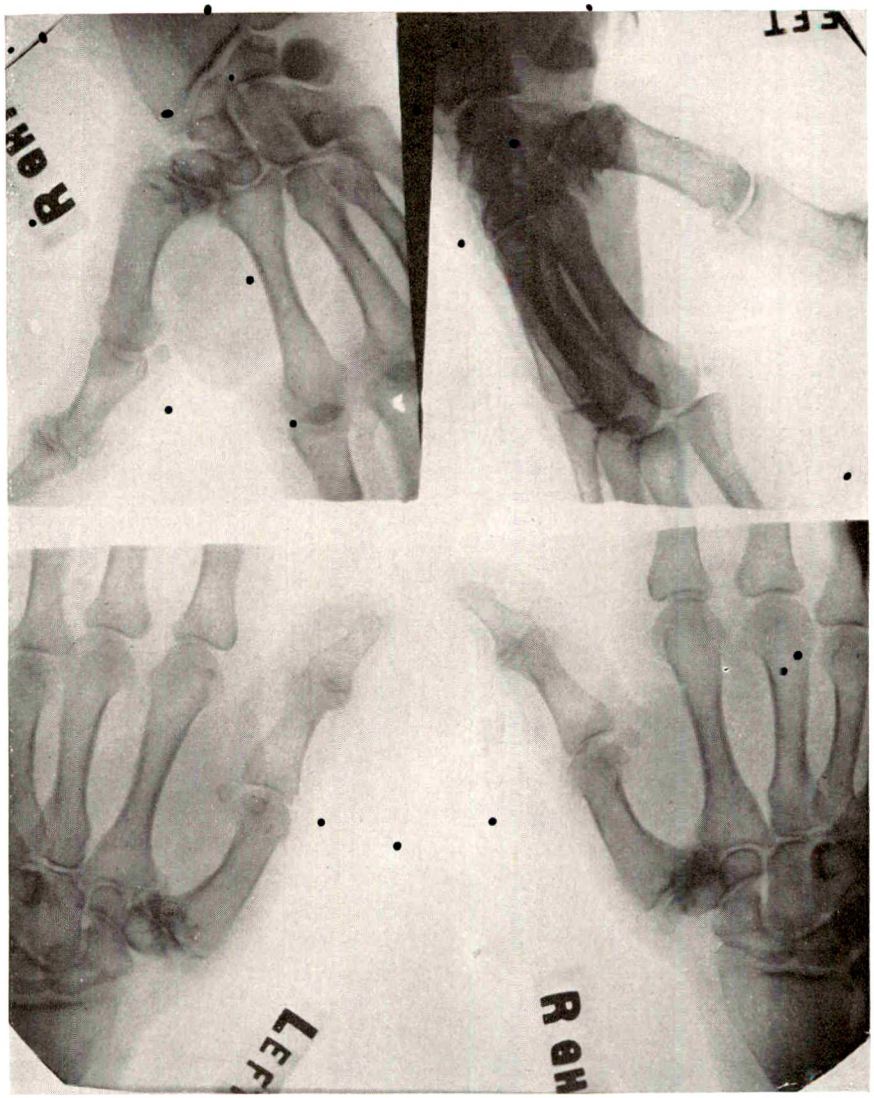


FIG. 17. The roentgenograms of a typical instance of injury of the carpometacarpal joint of the right thumb in older people. There is definite osteoarthritis of the carpometacarpal joint of each thumb with narrowing of the joint space and sclerosis of the adjacent articular borders. This sclerosis is more marked ulnarly than radially. Marked productive bony changes are seen on the radial side of the greater multangular bone. It is not possible to tell from the roentgenogram which is the side of symptomatology. This osteoarthritic joint may remain symptomless till axial trauma creates a painful joint or infraction fracture of this joint. It is not possible to prove the latter with certainty.

there was no gross evidence of fracture. This condition seems not uncommon, for we have later studied the roentgenograms of several other patients said to have this disability (Fig. 17).

Osteoarthritis may also be found along the radial half of the radial axis.

3. *Injury of the Lesser Multangular Bone.* Any injury of the lesser multangular bone is rare, since the bone is so well protected. A fracture dislocation of this bone was reported by Peterson. The type of trauma creating this disability was not described.

4. *Old Ununited Fracture of the Carpal Scaphoid.* The histories and roentgenograms of 5 cases of ununited fracture of the

carpal scaphoid were studied. All patients were males in the age group between twenty-two and thirty-seven. In every case, the fracture was situated at the middle of the bone. In 2 cases, there was displacement of the fragments, the distal fragment being rotated radially and proximally. Two cases showed moderate sclerosis of the ununited fragments. Irregularity in contour of the fragments was seen in only 1 case, and hypertrophic spurring of the fragment edges in 2 cases. In no case was arthritis of the wrist seen.

The time period between the injury and the roentgenograms examined varied between nine months and ten years. One man was roentgenographed for a recent Colles fracture and, in addition, showed an old ununited fracture of the scaphoid, which apparently was asymptomatic.

It is suggested that the scaphoid also be visualized in the lateral palmar flexion position, a position in which the bone is placed dorsovolarly. This view of the scaphoid, which may supplement the posteroanterior view in ulnar adduction, is only of use if enough palmar flexion is permitted. Hence, it may be of limited value in the differential diagnosis of fracture of the scaphoid.

5. *Fracture and Luxation of the Os Magnum.* The os magnum may be injured by a direct trauma, but this is not common. In an axial trauma, the capital part will act as a wedge, being driven between the scaphoid and the semilunar bone; or the head may break away, or capital dislocation take place at the cotyloid joint with the semilunar and the scaphoid. It may be expected that its capital part will more frequently be injured than its basal part, which is broad and firmly anchored.

Injuries of the os magnum are not common, since the bone is deeply placed and the trauma is passed proximally. Hence, it should not be unusual to find multiple fractures in association with fracture of the os magnum (Fig. 18).

6. *Basal Fracture of the Metacarpal*

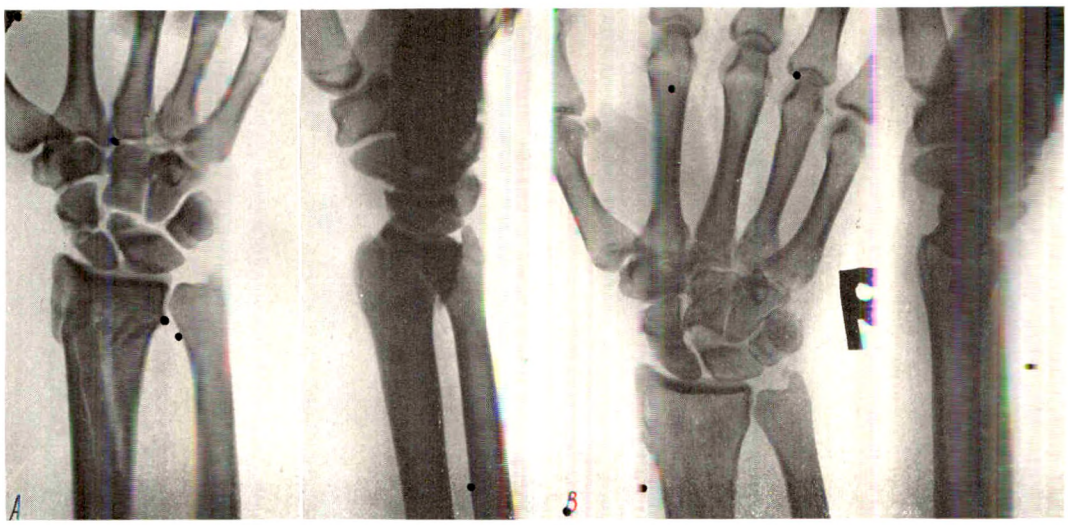


FIG. 18*A*. This patient, a male, aged nineteen, has a *multiple fracture disability of the radial axis*. There is seen a comminuted fracture of the lower end of the radius, a fracture of the scaphoid, and fracture (with rotation) of the caput of the os magnum. (Case of Dr. S. Ritchie of Kingston, N. Y., through whose kindness it is shown.)

FIG. 18*B*. The capital fragment of the os magnum was removed three and a half months after injury when the radial fracture and fracture of the scaphoid were healed in excellent position. There persists a probable dorsal displacement of the cuneiform.

Bones. Fracture of the base of a metacarpal bone, with the exception of the first, is relatively uncommon, the metacarpal bone usually breaking in its neck or shaft. Trauma is either direct or axial, greater damage being more probable by direct injury. The reverse is true, that fracture of



FIG. 19. This is a lateral roentgenogram of the palmarly flexed wrist of a man with painful dorsal prominence of the base of a metacarpal bone by axial trauma. Note the dorsal prominence, as if by bony hypertrophy, of the base of the second metacarpal bone, and the adjacent base of the os magnum. The prominence of the lesser multangular is not well seen in this reproduction. The scaphoid is placed dorsovolarly with prominence of its tuberosity anteriorly. This is the normal position of the scaphoid in lateral palmar flexion.

the neck or shaft of the first metacarpal bone is uncommon. Basal fracture seems more common in the more mobile fourth and fifth metacarpal bones. Dislocation of the metacarpal bases is rare. These statements are confirmed in a diagrammatic analysis of 700 metacarpal fractures by N. W. Roberts in the volume by Jones[4] (page 429).

6a. *Painful Dorsal Prominence of the Base of a Radial Metacarpal Bone by Axial Trauma.* We have seen 3 cases whose common characteristics place them in a group as a syndrome of axial trauma of the radial axis. In each case, a *painful* dorsal prominence developed at the base of a radial metacarpal bone, usually the second or third, at some undetermined time after an axial trauma to the knuckles. The deformity is seen best in palmar flexion of the wrist. The earliest development of the prominence seems to have been in the first week after injury.

It is probable that this bony prominence may be congenital, for we have seen it as a bilateral prominence in several normal hands. It may therefore have antedated any trauma, and become symptomatic by axial trauma.

In only 1 case was there a fracture of the metacarpal base—a chip fracture. In the other 2 cases, seen some time after injury, no fracture or evidence of old fracture was seen.

The etiology of such prominence, whether by direct or indirect trauma, by arthritic osteophyte or congenital enlargement may not always be evident. We are stressing a typical axial syndrome, which we separate from the common group, in all of which a basal dorsal metacarpal prominence exists.

The passage of force to the wrist may cause no fracture, but does create a strain of the joints through which the force passes, so that in all cases tenderness was present over the joint between the base of the metacarpal and the adjoining carpal bones, over the joint between the os magnum and the semilunar and scaphoid, and particularly over the wrist joint, which invariably showed slight limitation of motion. Proximal axial pressure in the line of the metacarpal confirms this tenderness.

The swelling at first may seem to be that of soft tissue but later there is bony hypertrophy, not only of the base of the metacarpal bone but, as in one case, of the adjacent lesser multangular. Recession of swelling can only be that of soft tissue and not of bone. The adjoining carpal bones, either the lesser multangular or base of the os magnum, seem to be placed as if in a depression, but this is relative because of the

bony prominence at the metacarpal base. The most serious late sequel which may make excision of the abnormal tuberosity necessary is the limitation of the use of a finger, as its extensor tendon subluxates over the bony prominence.

The best roentgenographic visualization of the bony prominence is the lateral view of the palmarly flexed wrist. The usual

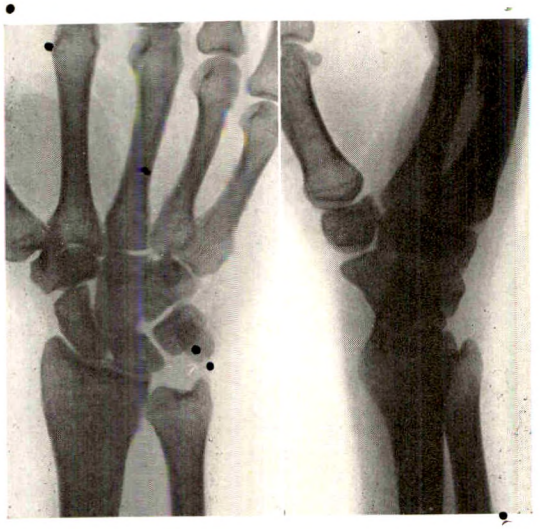


Fig. 20. Fracture of the pisiform bone is clearly indicated by the arrow.

anteroposterior and lateral views may show no abnormality (Fig. 19).

V. Fracture and Luxation of the Ulnar Carpal Bones

The disabilities of the ulnar carpal bones, *cuneiform*, *pisiform*, and *hamate*, are usually disabilities by direct trauma to the ulnar border of the hand (Fig. 20).

Axial trauma, though uncommon, may take place by a blow to the ulnar half of the hand as a fall on the ulnarly directed hand.

The reason such axial trauma is rare is that the bearing surface of the hypothenar eminence is small, and that the reception of any trauma by a fall on this part of the hand makes necessary an unnatural position of the hand. The resultant fracture is ordinarily simple, although there may be associated fracture or dislocation of the

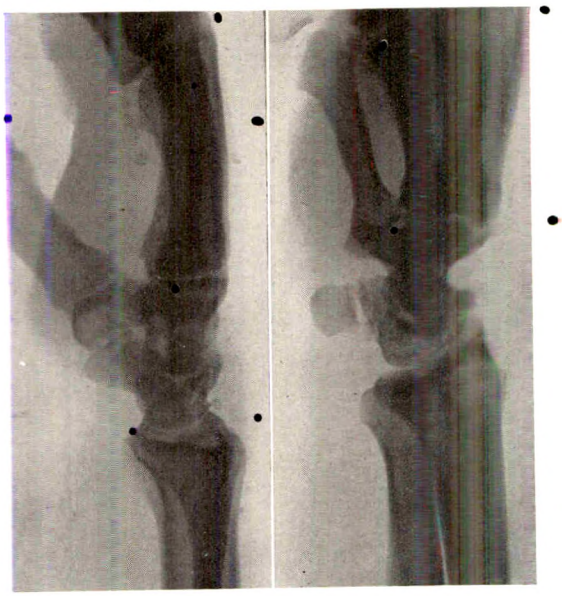


Fig. 21. Fracture by axial trauma causing a comminuted fracture of the lower end of the radius, a fracture of the cuneiform, and fracture of the second metacarpal head.

fourth and fifth metacarpal bones. Fracture of the cuneiform is sometimes associated with radial axis fracture.

The consideration of each fracture is unnecessary, but the mechanics of cuneiform

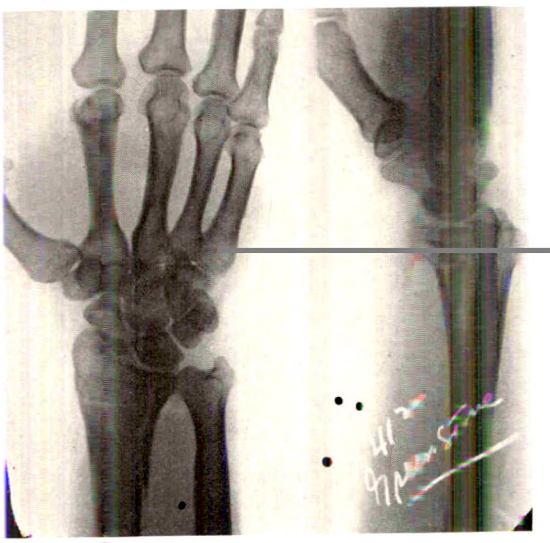


Fig. 22. Fracture of the cuneiform and of the dorsal aspect of the semilunar. The history in this case is not known, but it would seem to be a fracture by direct trauma.

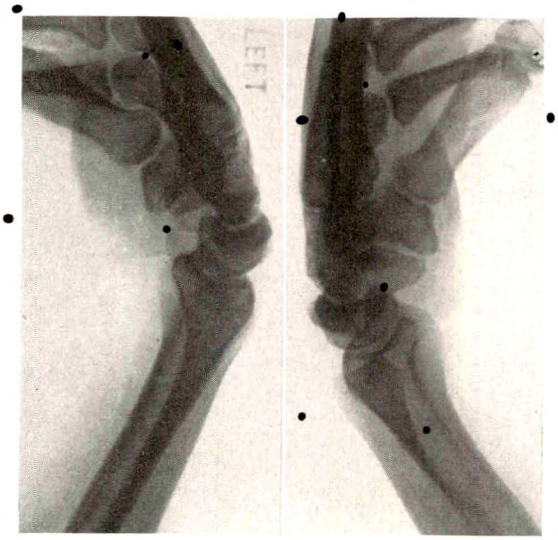


FIG. 23. Dorsal subluxation of the right cuneiform bone, seen only in the lateral view in palmar flexion.

fracture should be amplified. This fracture may be caused by both axial trauma and by direct injury to the volarly bent wrist. It is to be remembered that in palmar flexion of the wrist, the cuneiform bone becomes most dorsal, and hence vulnerable to direct trauma. It is a bone which occupies a position intermediate to both axes, and hence is seldom injured in radial axis injury. It is usually the proximal and ulnar part of the bone which is seen to be injured in the posteroanterior view, and the dorsal part in the lateral view. Fracture of this bone is not necessarily isolated, and may be combined with fracture of the lower end of the radius in both axial and direct injury. It was associated with fracture of the semilunar in one case (Fig. 21 and 22).



FIG. 24. Bilateral congenital fusion of semilunar and cuneiform bones was present, together with fracture of the scaphoid. The fracture is not well seen in this roentgenogram.

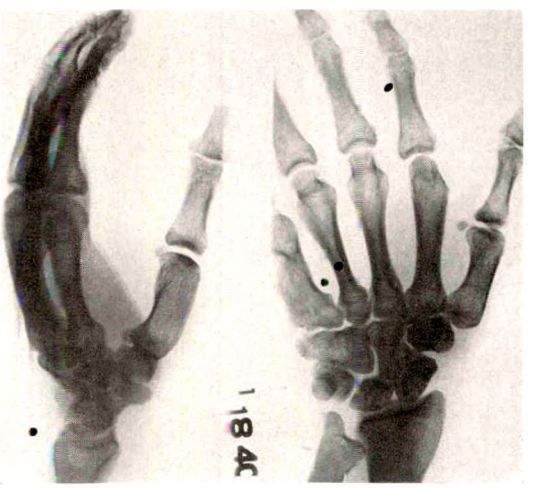


FIG. 25. A skeletal anomaly of the carpus, the most significant feature of which is a giant hamate bone, which articulates with a fifth metacarpal bone whose thickness is double the normal. The dumbbell-shaped metacarpal bones of the third and fourth fingers show a thin inner strongly accented cortical line. The first metacarpal is short.

Luxation of these bones is rarer than their fracture. The most probable is ulnar luxation of the pisiform since there is no bony obstacle to this luxation. Proximal luxation is possible when the torn tendon of insertion of the flexor carpi ulnaris carries the bone with it. Rotation of the bone would be most unusual.

The loosely jointed wrist may sometimes show ulnar luxation of the pisiform when the wrist is palmarly flexed and the flexor carpi ulnaris relaxed.

Dorsal subluxation of the cuneiform is rare (Fig. 23).

VI. OTHER INTERESTING CARPAL ENTITIES

As the many cases and their roentgenograms were examined over the long period of this study, certain interesting collateral observations were made.

These include congenital fusion of the carpal bones, especially of the semilunar and cuneiform bones, giant hamate bone, stencil atrophy of the carpus (Fig. 24, 25, 26 and 27), and vacuolization of the carpal



Fig. 26. Extreme subcartilaginous decalcification of carpal bones, so that a double cortical line is produced, which we call *stencil atrophy*, since the bones seem stencilled finely. A fracture of the shaft of the fifth metacarpal bone is present.

bones. Three syndromes are briefly described.

1. The *painful pisiform syndrome* is a syndrome in which the soft tissues over the pisiform and the base of the hypothenar eminence are swollen and tender, with or without abnormal mobility of the pisiform. The cause is minimal repeated trauma to the part. It should be differentiated from fibrositis, and from tendonitis with calcification of the flexor carpi ulnaris.

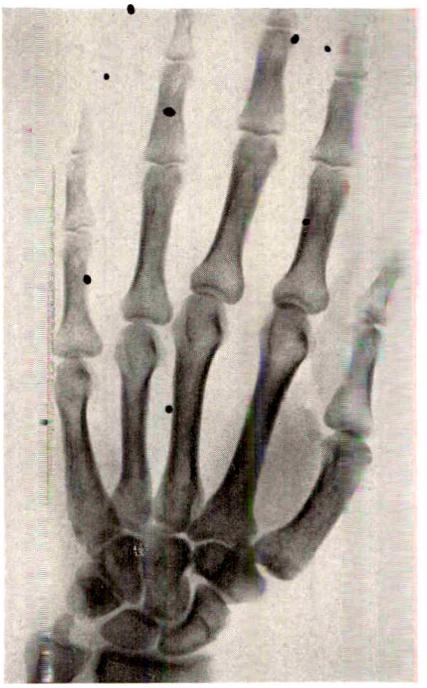


Fig. 27. Diffuse vacuolization of the carpal bones of the loosely jointed wrist. This is the left wrist joint of a woman, aged twenty-two, who made many hundreds of movements of pronation and supination of the forearm each day in working. Three years before she had fallen on the dorsum of the palmarly flexed wrist, with resultant dull pain in wrist, forearm and elbow. Wrist motion was free, but passive dorsovolar motion of the lower end of the ulna was much increased, a finding absent in the right wrist. The wrist was tender over the scaphoid and carpometacarpal joint of the thumb.

2. *Lateral Force Syndrome.* The ulnar corner of the base of the second metacarpal abuts against the inferoradial part of the os magnum. It seems reasonable, although we have no instance of it, that a lateral force applied to the second metacarpal may fracture not only the second metacarpal but also the corner of the os magnum (Fig. 28).

Lober reported a fracture of the outer border of the hamate, the size of a pea, which he believed had been caused by the impact of the fifth metacarpal against the hamate. A pressure fracture of the scaphoid was present. The patient's hand had been caught longitudinally in a sliding door and forced backward. This is an instance of a lateral force syndrome.

3. *The Carpal Canal Syndrome.* This syndrome is a median nerve neuritis and flexor tendonitis by compression of these structures in the bottleneck of the carpal canal, either by intrinsic or extrinsic cause. The typical extrinsic factor is the anteriorly

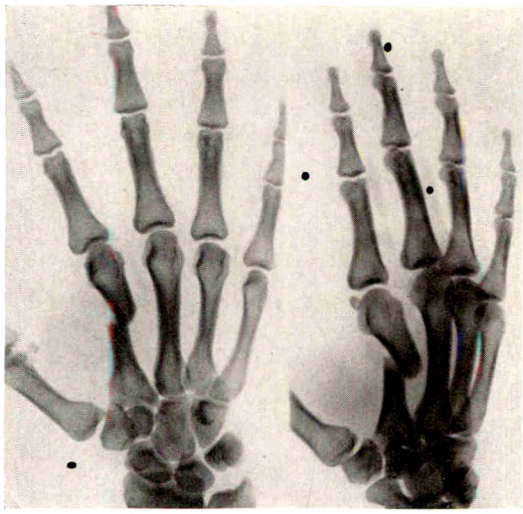


FIG. 28. Fracture of the shaft of the second metacarpal bone by lateral force. A sclerotic island in the center of an area of vacuolization was seen in the distal and radial part of the os magnum of each wrist.

dislocated semilunar bone. The intrinsic factor may be a direct trauma, squeezing or otherwise, to that part of the wrist, or, as is more likely, occupational. The rigid bony floor of the carpal canal and its ligamentous roof make it probable that any increase of tension, as by fluid, will adversely affect the contained structures, with the exception of the flexor longus pollicis tendon. Ordinarily, there should be easy release of this tension because of forearm space communication. It is for this reason that the intrinsic carpal canal syndrome is rare, but an occasional case may be met with in which release of pressure is not possible. One such case was seen.

CONCLUSION

Whether or not the principles and details contained within this paper are agreed with in part or wholly, one thing must be emphasized—that it is a certain mechanism of force which creates the shape and form of any fracture. It is the resultant type of fracture which is stressed in teaching and in textbooks rather than the creating force. There will be a better understanding of the fracture either as a single unit, or as a problem as a whole, if primary stress is laid on force conceptions.

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VOL. 51, NO. 4

TREATMENT OF CANCER OF THE LARYNX BY ROENTGEN IRRADIATION

A REPORT OF FIVE YEAR END-RESULTS*

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IT IS our purpose to present an analysis of our experiences and results in the treatment of cancer of the larynx by irradiation. This analysis includes all of the cases of larynx cancer, early and advanced, treated in the Department of Roentgenology and Radium Therapy of Temple University Hospital from 1931 to 1937 inclusive. It constitutes a five year survival study on a group of 36 cases.

Every patient in this series was first studied in the Chevalier Jackson Bronchoscopic Clinic of Temple University Hospital. In all cases the diagnosis of cancer was verified histologically. In each case the recognized indications for the various methods of treatment were carefully weighed. The treatment by irradiation in these patients was selected for one or several of the following reasons: (1) presence of metastasis in the neck on admission; (2) disease process too extensive for surgery; (3) suggested radiosensitivity of the tumor as determined by biopsy; (4) advanced age and poor operative risk; (5) complicating cardiac and renal disease; (6) recurrence after laryngofissure; (7) surgery refused by the patient.

ANATOMIC CLASSIFICATION

Inasmuch as tumors arising on vocal cords, ventricles, and the subglottic region have considerable similarity in their symptomatology, clinical course, and histopathology, we employ the anatomic classification as suggested by Martin.[4] We realize the disadvantage of such terms as "intrinsic" and "extrinsic"; nevertheless, their use in a limited capacity is permissible when the exact anatomic localization for each term is specifically designated. The term "cancer of the intrinsic larynx," as used in this paper, denotes origin of the tumor on the vocal cords, the ventricles, or the subglottic region, while "cancer of the extrinsic larynx" includes origin on the arytenoids, aryepiglottic folds, base of the epiglottis, and upper surface of the piriform sinuses. At times when one is dealing with a large, bulky tumor, it is impossible to determine the exact point of origin. The history, the histopathology, and the observations made during treatment, in which regression is carefully observed, may aid in making possible an accurate anatomic localization of the origin of the tumor.

INCIDENCE

Of the 36 cases, 23 (64 per cent) were classified as intrinsic, 13 (36 per cent) as extrinsic (Table 1). Twenty-nine of the cases were in the fifty to seventy year age group. The youngest was thirty-two and the oldest eighty-one years of age. Thirty of the lesions occurred in males, and 6 in females. In 53 per cent the lesions involved the right side of the larynx; in 31 per cent the left side, and in 16 per cent both sides were involved.

Cutler[2] emphasizes the importance of *fixation of the cord* in determining whether a particular lesion is curable by surgery or by irradiation. Unfortunately, our data on this point are incomplete and therefore inconclusive. It is our impression, however, that impairment of mobility itself is no contraindication to treatment by irradiation in intrinsic laryngeal cancer. In 11 of our intrinsic cases, there was definite fixation; 5 of these patients died of disease, while 5 are living and are well five or more

* From the Department of Roentgenology and Radium Therapy, Temple University Hospital, Philadelphia. Presented in part at the Twenty-sixth Annual Meeting, American Radium Society, Cleveland, Ohio, June 2–3, 1941.

TABLE I

CASES BY YEAR

	1931	1932	1933	1934	1935	1936	1937	Total
Intrinsic	0	0	4 (2)	3 (1)	4 (4)	6 (4)	6 (2)	23 (13)
Extrinsic	1	1 (1)	0	1	4 (1)	3	3 (1)	13 (3)
Total	1	1 (1)	4 (2)	4 (1)	8 (5)	9 (4)	9 (3)	36 (16)

Figures in parentheses indicate number of patients alive five years or more.

years after treatment. No impairment of the cords was recorded in 1 patient who is living and is free of disease. The 2 patients with cancer of the extrinsic larynx, in whom a definite statement of impaired mobility was made, have died of their disease.

During the past two years, we have made careful observations on impaired mobility and find that in many cases, except in those with extensive extrinsic laryngeal disease, as the tumor regresses and the tissues recover from the irradiation reaction, the cords become mobile, and only a slight degree of impairment due to scarring may persist. On the other hand, when the entire side of the larynx is fixed, or where there is definite cartilage invasion, the prognosis is less encouraging.

HISTOPATHOLOGY

The histopathology of the biopsies in practically all cases was squamous cell carcinoma. One of the cases was of a spindle cell, Grade 3, variety, while another was of the mixed type, showing areas of adenocarcinoma and squamous cell carcinoma (Table II).

Of the intrinsic group, 1 case was Grade 1, 13 cases were Grade 2, 7 were Grade 3, and 1 was Grade 4. Two of the Grade 3 cases are alive and free of disease after five years, while 9 of the 13 Grade 2 cases have survived five years or more. Grades 3 and 4 are infrequently seen in intrinsic larynx cancer. In explanation of the 7 cases recorded here, we wish to point out that these cases have been encountered and selected from a large series, and were referred for radiation therapy because of the anaplasia which suggested radiosensitivity. It has been frequently stated that squamous cell carcinoma, Grades 1 and 2, is relatively radioresistant.[4,9] Contrary to this, our findings indicate that these grades

TABLE II

HISTOPATHOLOGICAL TYPES AND GRADES

	No evidence of disease after 5 yr.		Dead as a result of disease		Dead from other causes*	Total
	Intrinsic	Extrinsic	Intrinsic	Extrinsic	Extrinsic	
Squamous carcinoma, Grade 1	1					1
Squamous carcinoma, Grade 2	9		[illegible]	2		15
Squamous carcinoma, Grade 3	2	2	[illegible]	5†	1	15
Squamous carcinoma, Grade 4	1	1		1		3
Spindle cell carcinoma, Grade 3			1			1
Not graded				1‡		1
	13	3	10	9	1	36

* Without evidence of recurrence or metastases from the cancer of the larynx.
† C.A. One case in which the histopathology was of a mixed type, showing areas of squamous cell and adenocarcinoma.
‡ H. S. Patient treated in 1931. Biopsy was not graded. Slide now not available for re-examination.

of tumor in the intrinsic larynx are quite radiosensitive. In our series the best results were obtained in the cases of histopathologic Grade 2 cancer.

METASTASES

Metastases are infrequent in cancer of the intrinsic larynx; only 4 of 23 cases (17 per cent) presented metastases on admission, and 2 cases developed metastases later in the course of the disease.

In 1 case there was a four year history of persistent hoarseness, and on endoscopic examination the entire right cord and ventricle and anterior third of left cord were found involved. Another patient had hoarseness of two years' duration, and had been treated by his local doctor for laryngitis. Biopsy revealed epidermoid spindle cell carcinoma, Grade 3. In a third case the metastases developed after treatment. This patient had a laryngofissure performed in October, 1932. Study of the tissue showed squamous cell carcinoma, Grade 3. Eight months after the laryngofissure a definite recurrence was found, and he was then given protracted fractionated, highly filtered external irradiation. A year later a second recurrence was verified histologically. The tumor grew quite rapidly, metastases occurred, and the patient died within one year. In all 3 patients, the metastases were treated by external irradiation alone.

One of the cases treated in 1937 developed a local recurrence and a metastasis to a cervical node two years after treatment. The biopsy from the recurrence and the aspiration biopsy of the metastasis both showed squamous cell carcinoma, Grade 3. A second full course of irradiation was given to the recurrence at the primary site including the metastasis in the field of irradiation. On completion of the roentgen treatment the metastasis was surgically exposed and interstitial radon implanted. This patient has had no further recurrence or metastasis and has survived now more than five and a half years since the original course of treatment.

In the extrinsic group, on the other hand, metastases were present on admission in 6 of 13 cases. The average length of survival in these 5 cases after treatment was twenty-two months. One patient, not included in this group, presented a mass in the left submaxillary region on admission. The laryngeal disease was located on the right side. As there is no histologic verification as to the nature of this mass, and inasmuch as he has been well and free of disease seven years after treatment, it must be assumed that it was not a metastasis.

It is very striking that none of the patients presenting proved cervical metastases on admission have survived. In the intrinsic group of patients without metastasis at any time during the course of the disease, the five year survival rate was 71 per cent. In a similar group of extrinsic cases, 3 out of 7 cases survived five years or more. It is evident that control of the primary disease and control of metastases are entirely different problems. None of the cases with metastases at the time of admission survived longer than three years.

External irradiation alone is insufficient for control of metastatic cervical disease. We must improve our methods of handling this particular group of cases. Our more recent experiences in a number of cases with cervical metastases falling into the intrinsic and extrinsic classifications indicate that cervical metastases may be controlled in a fair percentage of cases by combined treatment with external irradiation followed by surgical exposure and excision, if possible, and implantation of gold radon seeds. When sufficient time has elapsed, we will be in a position to compare the present results with those in patients whose metastases were treated more aggressively.

TECHNIQUE OF IRRADIATION

Physical factors of the radiating source employed in the series of cases herein reported are as follows: 180 kv. (average), constant potential, 5 ma., 50 cm. target-skin distance, and in all cases 2 mm. copper plus 1 mm. aluminum filtration. A daily

dose of 125 r, measured in air, was delivered through 10 by 10 cm. fields to each side of the neck at a rate of 5 to 6 r per minute. A total of at least 3,000 r was delivered to each skin field, necessitating a minimum of twenty-four consecutive days for the completion of treatment.

In our earlier cases the treatments were administered with the patient lying on the treatment table. In 1935 the advantage of the sitting position was called to our attention by one of our patients, an unusually intelligent woman, whose physical build rendered treatment in the horizontal position unusually difficult and uncomfortable. Since that date we have seated our patients in a chair fitted with a special immobilizing head rest (Fig. 1). This head rest is of value not only for its immobilizing effect but for maintenance of the desired degree of extension of the patient's head. In our experience the use of the sitting position and the above-described immobilization have added to the accuracy of our centering of the roentgen-ray beam, the maintenance of the patient's position during treatment as well as to the comfort of the patient.

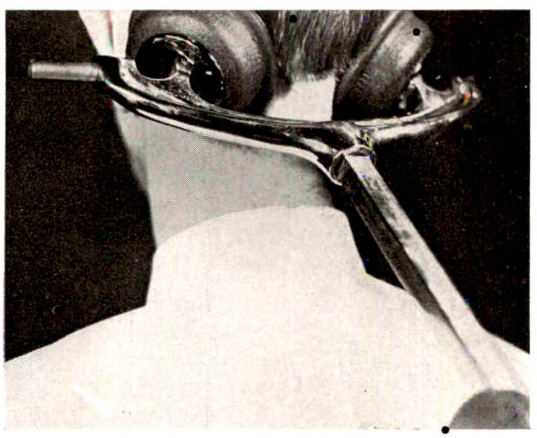


Fig. 1. The head rest as shown above is used to support the head during the treatment. It is fastened to the back of an ordinary sturdy chair by a set screw. By means of a swivel joint the arm between the chair clamp and head rest cushions can be extended and shortened or rotated in a vertical plane. The head rest cushions fit in the depressions below the mastoid and occipital prominences. With the flexibility of this apparatus any desired flexion or extension of head may be obtained and satisfactorily maintained during the treatment.

Up to 1936 a 10 by 10 cm. open field was used in practically all cases and occasionally fields as large as 12.5 by 12.5 cm. were employed. Such large fields necessarily produced extensive irradiation epidermitis which extended from the mandible down to the base of the neck, and a similarly extensive mucositis.

In 1936 the use of large rectangular fields was discontinued in favor of open circular fields of 7.5 cm. and 10 cm. diameter, and during the last three years interchangeable circular metal cylinders (cones) 5.5, 6, 7, 8, 9, and 10 cm. in diameter have been employed. With the smaller fields the cutaneous reaction may be as severe as with the large fields; however, the area and volume of tissue involved are much less. This has made possible the administration of larger tumor doses without producing severe local and generalized constitutional

Table III

Analysis of Metastases

	Number of cases	Number of 5 year survivals	Percentage of 5 year survivals
Intrinsic			
None on admission	19	13	69
None at any time	17	12	71
Present on admission	4	0	0
Metastases developed later	2	1	50
Extrinsic			
None on admission	7*	3	43
None at any time	7*	3	43
Present on admission	6	0	0
Metastases developed later	—	—	—

* Of these 7 cases, the first died after receiving four treatments; the second died of a heart attack nineteen months after treatment, showing no evidence of disease at the time of death; the third died with persistent disease ten months after treatment; the fourth died seven months after treatment; three are living and free of disease five and a half, seven and ten years respectively after treatment.

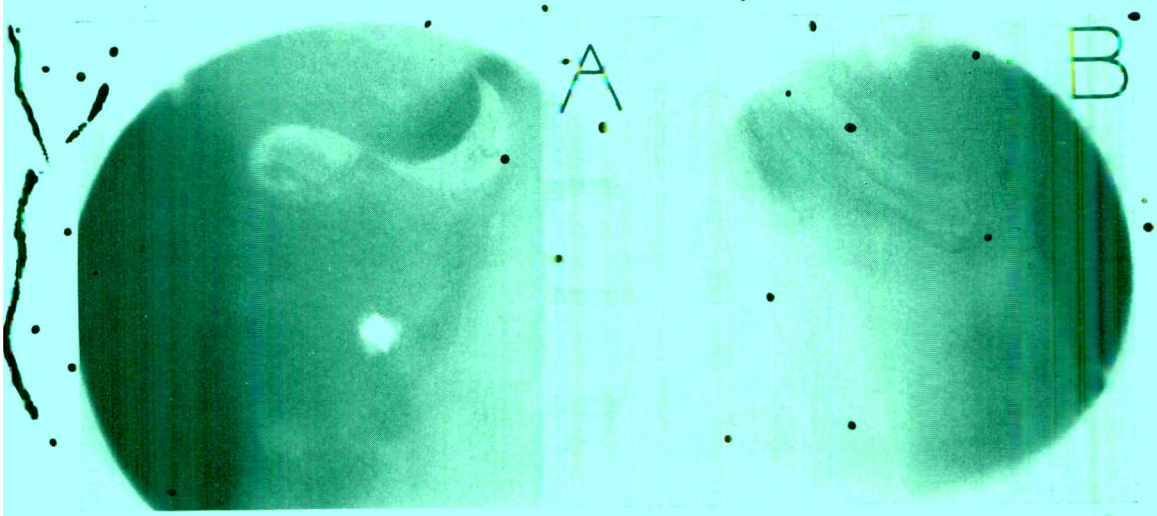


FIG. 2. These photographs are reproductions of the roentgenograms taken to check the accuracy of the centering of the radiation beam and the adequacy of the size of the portal. In *A* a cone 8 cm. in diameter was used and the lead marker, indicating the center of the beam, was centered slightly above the level of the cords for a supraglottic lesion. In *B* a 6 cm. diameter cone was used and centered at the level of the cords. In both cases the patient was set up exactly as for the treatment and the roentgenograms were exposed with the therapy machine.

reaction. When fields 10 by 10 cm. or larger are used in cases of extensive local or metastatic disease, it is with a great deal of concern that an air dose of more than 3,000 r to each side of the neck is given. The cutaneous and mucous membrane reaction that occurs is nearly always accompanied by pain, dysphagia, depression, and general debility. With the smaller fields, severe reactions are more localized, better tolerated, and even at the height of a severe mucositis the patient is able to take soft and liquid nourishment without the discomfort and pain that is experienced when the mucositis extends from the soft palate down to the base of the neck.

The introduction of cylinders or cones we believe is a refinement of irradiation technique. It must be emphasized that the use of cones requires accurate localization of the radiation beam. In all cases the anatomic location of the lesion should be carefully centered on the skin of the neck after



FIG. 3. In this method of checking the accuracy of centering, the approximate center of the lesion is carefully localized and marked on the skin. A wire loop the size of the cone to be used is placed accurately over the outlined skin portal. This is fastened in place with adhesive tape and the patient is then placed in the exact position in which the treatment is to be given. A lateral roentgenogram is exposed and an accurate check of the selected center and the adequacy of the size of field can then be determined.

thorough localization of the lesion, by study with a laryngeal mirror, by direct laryngoscopy when indicated, by external palpation and, for hypopharyngeal growths, by digital palpation of the lesion. The projected center and borders of the lesion are then marked on the skin, either with indelible dye or ink, or by tatooing with india ink.[5] The center of the skin field should always be localized in relation to such fixed anatomic landmarks as angle of jaw, hyoid bone, superior cornu of thyroid cartilage or thyrocricoid membrane. By correlating the position of the tumor with these anatomic regions, accurate centering of the skin field is assured. For each treatment set-up such careful localization is absolutely necessary (Fig. 2 and 3).

The selection of the size of cone is most important. For lesions of 2 cm. or less in diameter, as in cordal growths, or tumors involving only the tip of the epiglottis, a 5 cm. cylinder may be employed. For larger lesions or for lesions in other locations, especially where subglottic extension is suspected, the larger cones, 6, 7, 8, or 9 cm. in diameter should be used. We seldom use a cylinder smaller than 6 cm. in diameter except in the cases just mentioned. There are definite advantages in the use of cones over open portals. The end of the cone is placed against the skin of the patient, enabling very accurate centering of the beam. In addition, the contact of the skin with the end of the cone keeps the patient aware of the treatment and thereby helps him in maintaining his fixed position. With open portals, and if the head is not carefully immobilized, the slightest movement by the patient may entirely misdirect the beam.

For the cure of cancer by irradiation the

Table IV

TUMOR DOSE CALCULATIONS BASED ON 1,000 r, MEASURED IN AIR, AND DELIVERED TO EACH OF TWO LATERAL NECK FIELDS

(Tumor dose calculations corrected for back-scatter *

Irradiation Factors	180 kv. constant potential; 50 cm. target-skin distance; filter, 2 mm. Cu + 1 mm. Al				
Transverse Diameter of Neck	10 cm.	11 cm.	12 cm.	13 cm.	14 cm.
Size of Cone and Area	S-N† Distance 5 cm.	S-N† Distance 5.5 cm.	S-N† Distance 6 cm.	S-N† Distance 6.5 cm.	S-N† Distance 7 cm.
5.5 cm. (23.8 sq. cm.)	1192	1080	1000	920	850
6 cm. (28.3 sq. cm.)	1192	1080	1000	920	850
7 cm. (38.6 sq. cm.)	1280	1200	1110	1020	940
8 cm. (50.4 sq. cm.)	1380	1290	1200	1120	1040
9 cm. (63.8 sq. cm.)	1434	1340	1260	1160	1090
10 cm. (78.6 sq. cm.)	1488	1400	1370	1200	1120
10×10 cm. (100 sq. cm.)	1560	1470	1380	1300	1190

* Back-scatter and depth dose calculations are all based upon the tables and data of Quimby,[7] and Quimby, *et al.*[8]
† S-N indicates distance from skin to estimated center of neoplasm.

tumor dose delivered must be adequate. From Tables IV and V it is apparent that with the smaller cones larger dosages must be delivered to the neck fields. For example, in a neck of 10 cm. thickness, to deliver 8 threshold erythema doses to the tumor with 10 by 10 cm. portals, a dosage of about 4,500 r, measured in air, must be delivered to each of the two skin fields; for a 10 cm. diameter circular area the dosage would be 3,750 r; for a 7 cm. circular area, 4,000 r; for a 5.5 cm. circular area, 4,200 r.

TABLE V

COMPARATIVE ANALYSIS OF TUMOR DOSAGE FOR DIFFERENT SIZES OF SKIN PORTALS AND TUMOR DEPTHS

Size of Cone and Area	Transverse Diameter of Neck 10 cm. Skin to mid-point of larynx, distance—5 cm.						Transverse Diameter of Neck 14 cm. Skin to mid-point of larynx, distance—7 cm.					
	3000 r to each of 2 portals Tumor dose		4000 r to each of 2 portals Tumor Dose		5000 r to each of 2 portals Tumor dose		3000 r to each of 2 portals Tumor dose		4000 r to each of 2 portals Tumor dose		5000 r to each of 2 portals Tumor dose	
	r	T.E.D.*	r	T.E.D.	r	T.E.D.	r	T.E.D.	r	T.E.D.	r	T.E.D.
5.5 cm. (23.8 sq. cm.)	3570	5¾	4758	7¾	5960	9½	2530	4¼	3400	5½	4250	7
6 cm. (28.3 sq. cm.)	3570	5¾	4768	7¾	5960	9½	2530	4¼	3400	5½	4250	7
7 cm. (38.6 sq. cm.)	3840	6	5120	8	6400	10	2530	4½	3760	6	4700	7½
8 cm. (50.4 sq. cm.)	4140	6¼	5520	8½	6900	10½	3120	4¾	4160	6¼	5200	8
9 cm. (63.8 sq. cm.)	4302	6½	5736	8½	7170	10¾	3270	4¾	4360	6½	5450	8½
10 cm. (78.6 sq. cm.)	4464	6½	5952	8¾	7440	11	3360	5	4480	6¾	5600	8½
10×10 cm. (100 sq. cm.)	4680	6¾	6240	9¼	7800	11½	3570	5¼	4760	7	5950	8½

* Threshold erythema dose.

COMPLICATIONS OF RADIATION TREATMENT

The cutaneous reaction is a necessary result of radiation treatment. The typical skin reaction begins with a slight erythema which usually makes its appearance between the tenth and the fourteenth day of treatment. Gradually the erythema assumes a deeper red color and dries. During this stage it may present a definite leathery appearance. When a dosage of 3,000 to 3,500 r (depending on the size of field used) is administered to each of the two fields, blistering and desquamation begin to appear. In the majority of cases, within a period of about five days after completion of treatment, there is a very marked superficial ulceration which, with proper surgical care, goes on to complete healing within a period of ten to fourteen days. It is important to care for the irradiation reaction as one would in any surgical case of severe burn. If the ulceration is allowed to dry it becomes crusted and infected, and bleeds on the slightest manipulation. Such a condition is favorable to extensive infection, necrosis of the superficial layers and persistent ulceration (Fig. 4).

In the surgical care of these reactions, we have found the following procedure very efficacious. The raw surface is cleansed with dilute hydrogen peroxide and sterile distilled water, and then gently sponged with

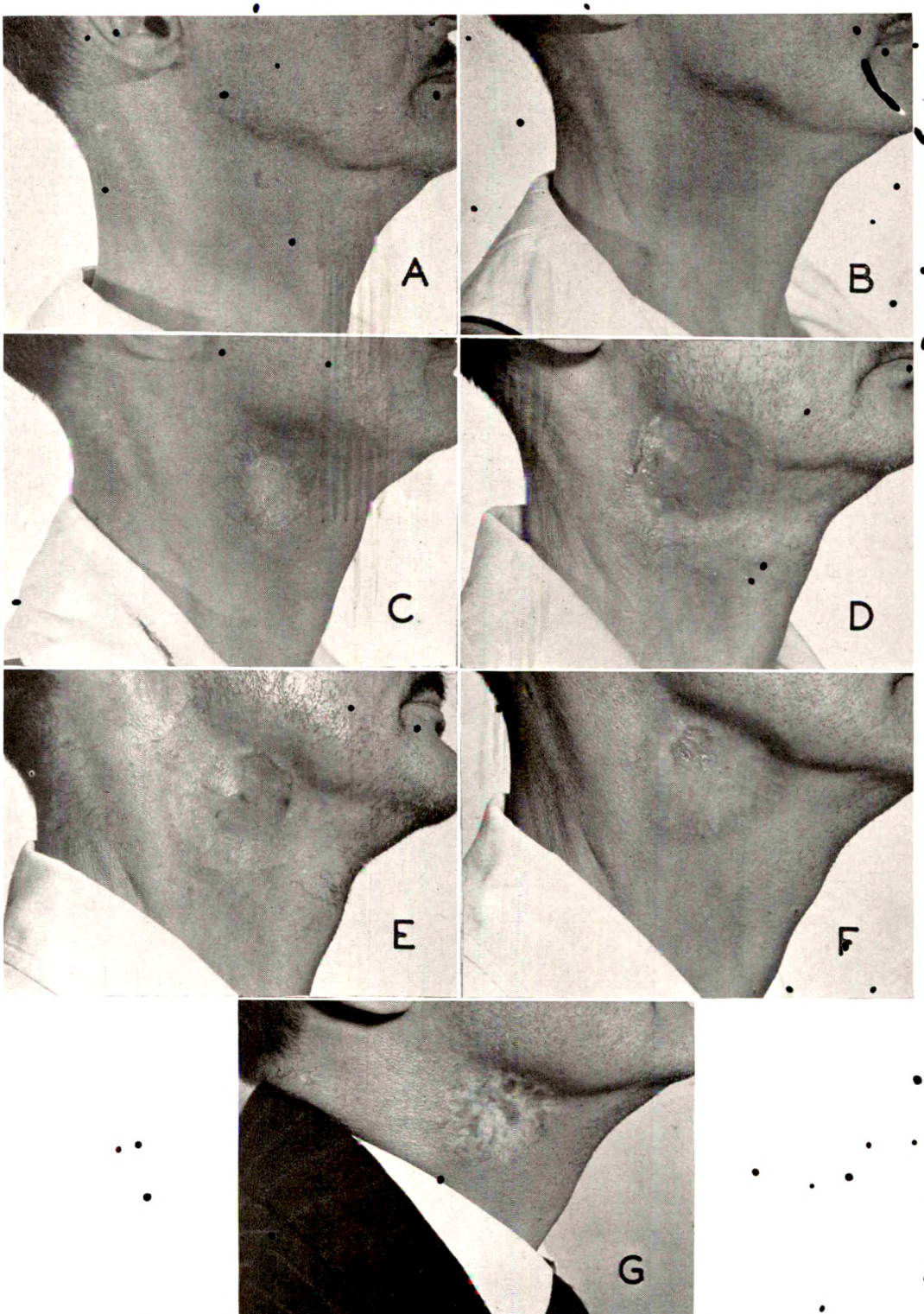


Fig. 4. (See next page for description.)

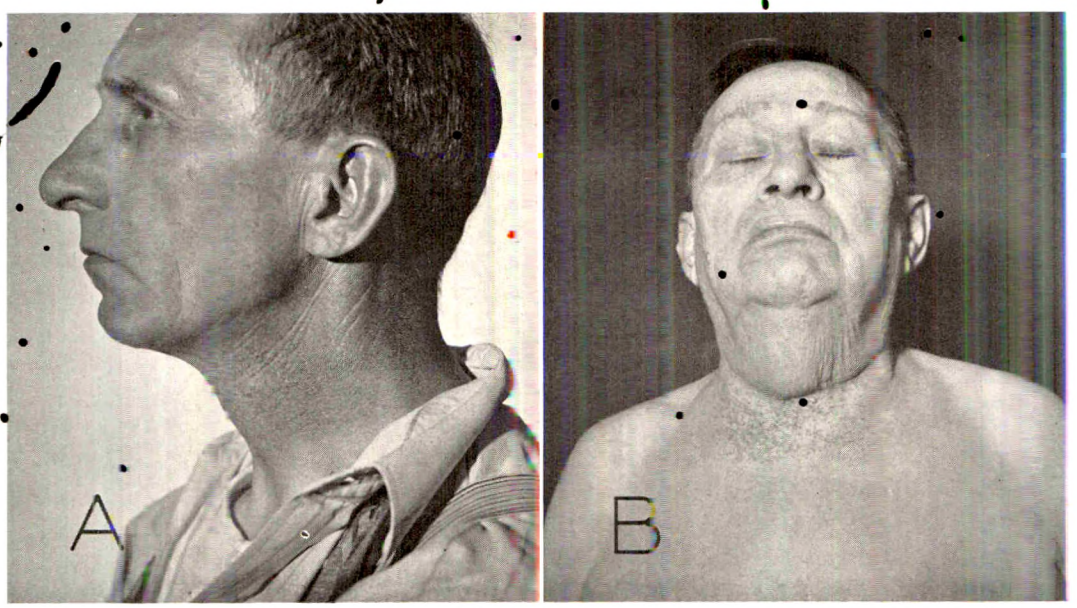


FIG. 5. *A* (L.L.) Appearance of skin of neck five years after treatment. This patient received a total of 3,000 r to each side of the neck through 10 cm. diameter open portals in twenty-four consecutive days. This is a minimal amount of skin change.

B (L.B.) This patient presents considerably more skin atrophy, telangiectasia and submental edema than the patient in *A*. In 1932 this patient received thirty-six consecutive daily treatments to each side of the neck through 10.5 by 10.5 cm. fields, equivalent to a total of 3,600 r to each field. In November, 1936, a proved recurrence was treated. Twenty-four consecutive treatments were given through 7.5 cm. diameter open portals for a total of 3,000 r to each portal. This photograph was taken ten years after the first treatment series and six years after the second.

mineral oil. Gauze cut into desired shapes and sizes, either rectangular or circular, is heavily impregnated or overspread with an abundance of boric acid ointment. This sterile gauze is placed over the entire area of irradiation reaction. A dry dressing is fastened in place over this. (The application of sulfathiazole powder alone is not advised as the powder dries and crusts, producing more suppuration, fissuring of the wound and greater discomfort to the patient.) These dressings are changed at least once or twice a day. With this care the patients have been very comfortable, and in most cases have been able to carry on their regular daily routines. We have not observed any complications. Complete healing occurs in a period of ten to fourteen days, leaving a soft, pliable, and pale red skin. Gradually the redness blanches and the skin then begins to show varying degrees of pigmentation. Eventually the pigmentation disappears and telangiectasia appears (Fig. 5).

FIG. 4. A series of photographs showing the appearance of the skin during the course of treatment and after healing. *A*, nineteenth day of treatment, total of 4,750 r to each side of the neck. Treatment was completed on twentieth day at which time a total of 5,000 r was delivered to each side of the neck, through a cone 5 cm. in diameter. *B*, appearance of erythema four days after completion of treatment. *C*, fifteen days after completion, there is definite blistering and central ulceration. *D*, twenty-two days after completion, ulceration is severe although healing along the periphery of the field has taken place. *E*, twenty-six days after completion, healing progressing. *F*, fifty-seven days after completion of treatment, ulceration completely healed, although the newly formed skin traumatizes easily as shown above. *G*, the appearance of the skin nineteen months after completion of treatment.

The mucosal reaction usually appears after the skin reaction has become definite. If a dosage of 3,500 to 4,000 r is delivered to each skin portal, the acute mucosal reaction begins to appear. The early visible manifestation of reaction in the mucous membrane is a reddening which may be accompanied at times by lymphedema. Along with this the patient begins to complain of a "scratching sensation in the throat," or a "sticking" or "lumpy" sensation. Later, as the reaction becomes more intense, the act of swallowing is progressively more difficult. The formation of the whitish membrane or coating over the laryngeal mucous membrane is comparable to the ulcerative stage of the irradiation reaction in the skin. It is interesting to note that even though the acute form of the mucositis appears later than the skin ulceration it is of shorter duration.

Early in the development of the mucous membrane reaction, an increased amount of mucus is formed. Frequently this is accompanied by spasmodic coughing. As the mucositis abates, the viscidity of the secretion is lessened and the symptoms of "hawking" and spasmodic cough disappear. Treatment directed at cleansing the mouth and dissolving oral and hypopharyngeal secretions aids greatly in alleviating discomfort during this stage of the reaction. Irrigations carried out at intervals of from two to three hours with a solution of sodium bicarbonate and salt or alcaroid in lukewarm water are quite helpful. At night the patient is instructed to carry out the irrigation only when awake.

Dyspnea is seldom observed except in cases of bulky supraglottic tumors or advanced disease. In this series of cases, it was necessary to perform a tracheotomy in 4 cases shortly after treatment. All 4 patients died. In 1 case, two months after the completion of treatment, the patient died of strangulation because of refusal to permit a tracheotomy. At autopsy the strangulation was found to be due to stenosis of the larynx by residual cancer and lymphedema. In 5 patients a tracheotomy was performed before the institution of treatment, and 2 of these patients are living and well five years or more after treatment. In another case, it was necessary to perform a tracheotomy five days after beginning treatment because of marked dyspnea. This patient died five days later of a diffuse cellulitis originating in the tracheotomy wound. As a result of this and other tragic experiences, we are extremely pessimistic whenever tracheotomy becomes necessary *during the course of irradiation*. We have a rule which we try very hard to live up to; namely, *whenever our preliminary studies suggest that obstruction may develop, tracheotomy is done immediately and irradiation is not started until healing is complete*. To disobey this rule is to court fatal complications.

Dysphagia, which occurs at the height of the irradiation reaction, is of limited importance in intrinsic laryngeal cancer. When the cancer is in the region of the arytenoids, dysphagia may be quite pronounced, because the reaction is usually most intense at the tumor site. With a bulky lesion, the difficulty in swallowing is naturally greater. Lymphedema of the arytenoids, aryepiglottic folds, and piriform sinuses is not infrequently observed and when it occurs, there may be marked mechanical dysphagia. In our experience, we have observed this association not only during the treatment but also weeks and months after completion of treatment (Fig. 6). Anesthesin lozenges or powders will relieve soreness when it is due to reaction at the base of the tongue.* When dysphagia interferes with nutrition, nasal tube feedings may be instituted. During the acute stage of the mucous membrane reaction, and especially in the presence of lymphedema of the arytenoids and the piriform sinuses or where bulky disease is

* A powder which was suggested to us by Dr. F. L. Follweiler, and which we have employed with excellent results, may be compounded as follows: codeine phosphate gr. ½; benzocaine, gr. 1; acetphenetidin, gr. 2; acetylsalicyclic acid (pulv.), gr. 2. A half or a full chartula is placed on the back of the tongue and allowed to dissolve fifteen minutes before eating. This produces a superficial anesthesia which allows the patient to swallow soft and liquid foods with very little discomfort.

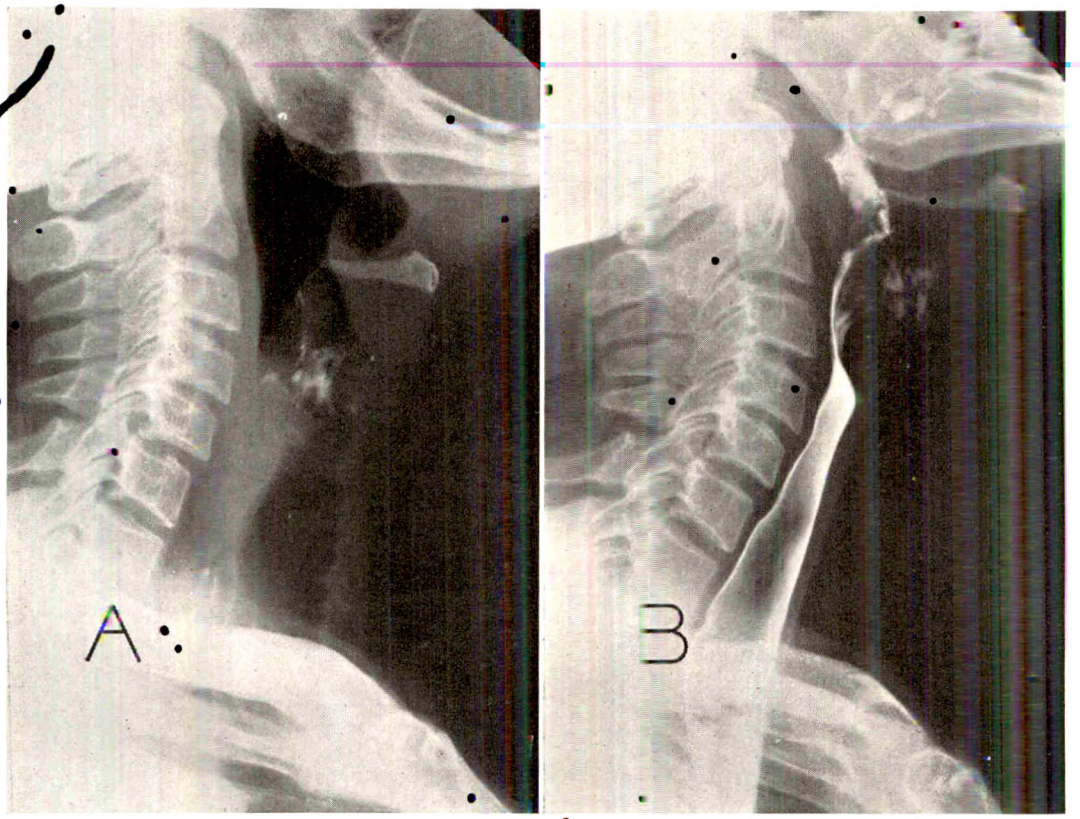


FIG. 6. *A* and *B*, lateral roentgenograms demonstrating a marked degree of edema of the arytenoids and the postcricoid space. In *B*, encroachment on the upper end of the esophageal lumen is revealed by barium. This patient had a squamous cell carcinoma, Grade 2, involving one cord and was treated in December, 1939. These roentgenograms were taken in March, 1940. The edema persisted for approximately one year and then gradually subsided.

present, utmost care should be observed when using a feeding tube that remains in place for several days. Sloughing of the hypopharyngeal or crico-esophageal mucous membrane may occur due to pressure necrosis. Nasal tube feeding formulae should be adequate in caloric content and should include vitamins and pureed vegetables. It is rarely necessary to resort to parenteral feeding.

Radionecrosis is infrequently observed with roentgen therapy alone, being more commonly encountered when supplementary interstitial irradiation is used. In the presence of severe infections, however, radionecrosis may occur. When tumor has invaded cartilage the ensuing chondritis usually results in sloughing. We have observed this in one patient who is now free of disease five years after treatment. He received a total dosage of 3,125 r to each side of the neck through 12.5 by 12.5 cm. fields, and on completion of the treatment he developed repeated attacks of erysipelas of the skin of the face and neck, prolonging and severely complicating the cutaneous reaction. Approximately four to six months later, sloughing of the laryngeal structures occurred with extrusion of cartilage and production of laryngeal obstruction, necessitating a tracheotomy. The sloughing and extrusion continued for a period of about three years and ended in complete loss of all laryngeal cartilages. This is the only case in the series in which this has occurred. Occasionally in cases necessitating re-

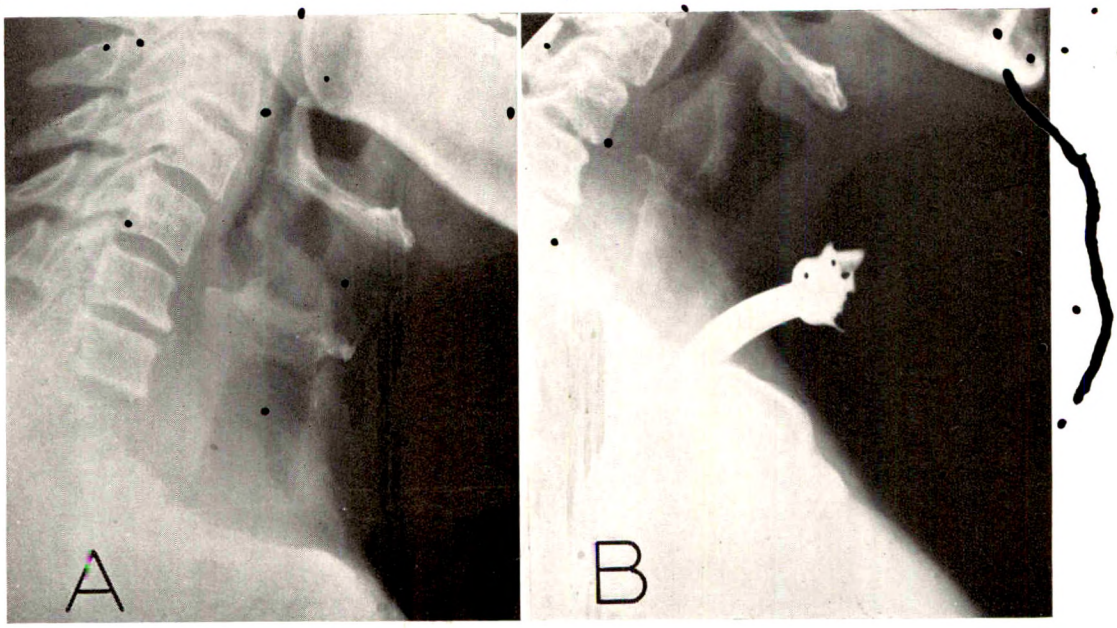


Fig. 7. These roentgenograms illustrate the rare complication of radionecrosis and sloughing of the laryngeal cartilages. This patient had an extensive squamous cell carcinoma, Grade 3. He was given treatment through two lateral neck fields, 8 cm. diameter cones, 3,875 r total to each field. Seven months later recurrent laryngeal disease and cervical metastases were proved. A second course of treatment was given. *A* shows the appearance of the larynx prior to the first treatment series. In *B* sixteen months later, a great portion of the laryngeal cartilage had been destroyed and extruded as is shown by the air space.

treatment, a varying degree of superficial necrosis of tumor on the cord or ventricular band is observed (Fig. 7).

Hemorrhage has not occurred in any of the cases in this series. Recently we have observed one patient in whom a fatal hemorrhage resulted during the course of treatment. As no postmortem examination was obtained, the cause of this hemorrhage was not determined. It is believed, however, that in all probability the tumor had eroded one of the larger laryngeal or pharyngeal vessels.

Xerostomia, the drying of salivary secretion, may be produced when large fields, which include the submaxillary and parotid regions, are used. This complication has been observed in a number of patients, although in those cases receiving radiation through a small field it was not encountered. In a number of cases, the decrease in the production of saliva resulted in the accumulation of inspissated secretions, which caused symptoms of hawking, discomfort in the throat, and, in 2 cases, persistent hoarseness. In the latter 2 cases, the secretions were found overlying the cords and on the upper ventricular membranes. Oral irrigations with bicarbonate of soda and applications of gomenol to the mucous membranes at frequent intervals gradually relieve this condition. When the secretory glandular function has not been completely destroyed, it is possible to stimulate secretions by chewing gum or lemon peel, and by massaging over the glands.

Submental lymphedema is not infrequently observed and except for the resulting disfigurement is of no consequence. Immediately after treatment lymphedema may be quite marked; it may continue intermittently and to a varying degree for months or possibly years.

Voice after Irradiation. We fully realize that voice to a great many people is indispensable and economically may be of primary importance. Nevertheless, we wish to emphasize that the restoration of voice

is merely of incidental importance and should not be a major consideration in the selection of the treatment method for cancer of the larynx.

During the height of the irradiation mucositis all patients develop a varying degree of hoarseness and not infrequently some become temporarily aphonic. As the irradiation mucositis subsides the voice begins to return and after complete healing it may return to its original quality. In some cases, there may be so slight a change in pitch that it is unnoticed by the patient or his friends.

In one of our patients treated in 1935, now a seven year survival, free of disease, complete aphonia had been present for more than twelve years before the institution of roentgen treatment. During those years the constant laryngoscopic finding was that of fungating carcinoma, though repeated biopsies resulted in identification of the desmoplastic reaction and histologic interpretation of the pathologic process as a "fibroangioma." Finally a biopsy in 1935 showed a definite squamous cell carcinoma, Grade 2. Following roentgen treatment the voice gradually improved and now is of excellent quality and carrying power.

Where the disease is extensive and destruction of the cords has occurred prior to the treatment, definite impairment of voice is an unavoidable sequela. The degree of impairment depends on the amount of destruction. In some cases, there is an actual narrowing of the glottic chink due to marked cicatrization because of extensive destruction of the cords, especially when the disease is in the region of the anterior commissure.

When it becomes necessary to repeat a course of treatment for a recurrence, a very marked impairment of voice is to be expected. Both the quality of voice and its carrying power may be greatly impaired. In these cases the voice may be barely more than an audible whisper.

RESULTS OF TREATMENT

Reports on one method of treatment alone are of necessity based on a selected group of cases. We wish to emphasize that studies and reports of end-results in cancer, of whatever anatomic region, should be based on *all cases seen* regardless of the extent of disease or method of treatment. Nevertheless, an analysis of results in such a selected group yields important data on the efficacy of the particular method of treating the various stages and anatomic varieties of the disease.

As for cancer in general, treatment of laryngeal cancer may be by surgery, by irradiation, or by a combination of the two methods. Each procedure has certain indications and advantages, and therefore a place in the scheme of treatment. *These methods of treatment are not competitive. That particular method should be selected which, in the individual case, offers the patient the greatest chance of survival.* While emphasizing their non-competitive status, we feel that an analysis of the results of an individual treatment method is permissible for it may help in defining the indications used in the selection of the particular treatment modality.

Table VI epitomizes the results in this series of cases. Of the 23 cases classified as intrinsic, 9 died of the disease. Thirteen have been free of disease five years or more. This constitutes a five year survival of 59 per cent. Of the 13 cases classified as extrinsic, 9 died of cancer. One died nineteen months after treatment of heart disease without evidence of cancer, either recurrent or metastatic. Only 3 patients remain living and free of disease five or more years after treatment, for a five year survival rate of 25 per cent.

RECURRENCES

In the intrinsic group there were 4 recurrences. In the first case a laryngofissure was performed for an ulcerating squamous cell carcinoma, Grade 4, involving the anterior two-thirds of the left cord. Eight months later a recurrence was verified histologically. A full course of roentgen therapy, 7,200 r to each side of the neck, was

TABLE VI

FIVE YEAR END-RESULTS
1931 to 1937 Inclusive

	Intrinsic Cancer	Extrinsic Cancer
Total Number of Patients	23	13
Indeterminate Group		
A. Dead as a result of other causes without recurrence or metastases	1*	1*
B. Lost track of without recurrence	0	0
Total number of indeterminate group	1	1
Determinate Group		
Total number of cases minus those of indeterminate group	22	12
Failures of Treatment		
Dead as result of cancer	9	9
Successful Results		
Free of disease after five years or more	13	3
Net Five Year End-Results (successful results divided by determinate group)	(13/22) 59%	(3/12) 25%

* These 2 cases are classified indeterminate inasmuch as these patients died a year after treatment of natural causes other than cancer and at the time of death, there was no evidence of recurrence or metastasis. These cases are of no value in determining the efficacy of a treatment method and therefore are not considered in the statistical end-result evaluation.

given through 10 by 10 cm. fields. Ten months later, a second recurrence was noted and a laryngectomy was advised but refused by the patient. A tracheotomy was performed and roentgen therapy was administered, 75 r daily through a 7.5 by 7.5 cm. anterior neck field, for a total dose of only 900 r. This patient died fifteen months later of local and metastatic disease.

The second case was a squamous cell carcinoma, Grade 3, recurring fourteen months after a full course of roentgen treatment consisting of 3,500 r total to each side of the neck through 7.5 cm. circular fields. The biopsy was positive. A second course of roentgen therapy was not given immediately. Nineteen months later roentgen therapy was started but the patient died of a lobar pneumonia during the course of treatment.

A third case developed a recurrence eight years and two months after commencement of first treatment series. At the time of recurrence, November, 1941, he was eighty-two years of age and a biopsy revealed a squamous cell carcinoma, Grade 2. This patient received a second course of roentgen therapy in December, 1941, and at the last examination in November, 1942, there was no evidence of recurrent disease.

In the fourth case a recurrence and a metastasis to a mid-cervical node was varified histologically two years after the original treatment of 3,125 r total to each side of the neck for a squamous cell carcinoma, Grade 3. A second course of treatment consisting of 3,875 r to each side of the neck was given. In addition to this external irradiation, the metastatic node was surgically exposed and radon seeds were implanted. This patient has remained alive over five years after the original treatment and up to the present time is free of further recurrence and metastases.

Of the extrinsic group 9 died of laryngeal disease. The question of recurrence is difficult of evaluation as 6 of these patients had metastases on admission, while 2 started treatment but died before completion. The average survival for the group completing treatment (7 cases) was eighteen months.

SUMMARY

This paper presents an analysis and a five year survival study of all cases of cancer of the larynx treated at Temple University Hospital Roentgen and Radium Therapy Department between 1931 and 1937. The technique of roentgen treatment and its complications are fully discussed.

An arbitrary classification which recognizes two distinct varieties of laryngeal cancer is used. So-called intrinsic cancer is predominantly of squamous cell, Grade 2, variety, whereas the extrinsic is usually of Grade 3 or 4. Squamous cell carcinoma, Grade 2, is a radiosensitive growth when limited to the intrinsic larynx. It is of

interest to note that of the 4 recurrences in the intrinsic group 3 were Grade 3 and 1 was Grade 2.

Metastases are infrequently observed in intrinsic laryngeal cancer. Of our cases which did not develop metastases at any time, 71 per cent survived more than five years. Of the extrinsic group 46 per cent had metastases on admission and none of these patients survived.

For the intrinsic group treated by roentgen irradiation, the net five year survival without evidence of recurrent or metastatic disease is 59 per cent, while for the extrinsic group it is 25 per cent.

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APRIL, 1944

IRRADIATION SICKNESS*

A HYPOTHESIS CONCERNING THE BASIC MECHANISM AND A STUDY OF THE THERAPEUTIC EFFECT OF AMPHETAMINE AND DEXTRO-DESOXYEPHEDRINE

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IRRADIATION sickness is defined as a feeling of sickness, with acute general symptoms, which sometimes follows the application of massive doses of roentgen and radium rays. The present study is limited to the use of roentgen rays. The syndrome of irradiation sickness may include variable degrees of weakness, exhaustion, lassitude, anorexia, nausea, and vomiting. Manifold explanations of the etiology of irradiation sickness include (*a*) the effect of gases produced by the high tension electric field; (*b*) the effect of the electric field surrounding the patient; (*c*) chemical changes produced in the body; (*d*) absorption of products of cell destruction; (*e*) sympathetic-parasympathetic nervous system imbalance; (*f*) injury of the suprarenal glands; (*g*) injury of sympathetic or parasympathetic nerves or nerve endings; (*h*) nervous strain or psychic influences, and (*i*) destruction of intestinal mucosa.

None of these theories has offered a comprehensible explanation of the basic mechanism. Therapeutic measures are varied and controversial. Steinberg[5] recently reviewed the medical literature and believes that no therapeutic measure is specific and found that the recently advocated use of thiamine chloride had little effect in either the prophylaxis or treatment of radiation sickness. Whitmore[7] reported the favorable effect of thiamine chloride combined in some instances with concentrated liver extract and calcium salts. A detailed review of the diverse methods and therapeutic agents used in the treatment of irradiation sickness may be found in the aforementioned and other articles, and will not be reiterated.

CLINICAL EFFECT OF AMPHETAMINE AND DEXTRO-DESOXYEPHEDRINE ON IRRADIATION SICKNESS

The study of the present series of patients with irradiation sickness was initiated because of the prevalence of physical incapacity among patients receiving intensive roentgen therapy despite accepted therapeutic measures. Ambulatory patients receiving prophylactic irradiation of the trunk regularly manifested marked weakness, lassitude, nausea, and occasional vomiting. Interference with occupational or other activities often was undesirable even though interruption of irradiation was not required by the severity of symptoms. The favorable effect of amphetamine† on various asthenic states suggested possible benefit in irradiation sickness. The use of amphetamine was also suggested by its effectiveness in the prophylaxis and treatment of symptoms of seasickness, symptoms which some patients described as very similar to irradiation sickness. An inhibiting effect on the gastrointestinal tract indicated possible benefit to the nausea and vomiting. Dextro-desoxyephedrine‡ was studied because of its similarities to amphetamine. Evaluation of the relative effectiveness of the two drugs was not attempted.

The present study was limited to the therapeutic effect of amphetamine and dextro-desoxyephedrine on irradiation sickness and excludes the possible prophylactic effect. Neither drug was administered until patients demonstrated definite symp-

† Benzedrine sulfate brand of Amphetamine manufactured by Smith, Kline and French Laboratories, Philadelphia, Pennsylvania, used in this study.

‡ A new drug, limited by Federal Law to investigational use, supplied through courtesy of Abbott Laboratories, North Chicago, Illinois.

* Presented at the meeting of the Michigan State Medical Society, Detroit, Mich., Sept. 22–25, 1943.

toms of irradiation sickness during the course of roentgen therapy. The drugs were seldom introduced until patients complained of severe weakness, considerable nausea and occasional vomiting. A few patients received other medications from referring physicians. These medications were maintained inasmuch as their failure in preventing irradiation sickness implied little confusion in evaluation of the therapeutic effect of amphetamine or dextro-desoxyephedrine.

Accurate evaluation of any therapeutic agent in irradiation sickness is difficult and must be based on subjective symptoms of the patient. In the present series, the patient's general physical condition prior to the course of roentgen therapy was noted. The condition was designated "good" when the patient was able to carry on normal activities; "fair" indicated partial limitation of activities or a semi-invalid; whereas "poor" or "terminal" designated bed-ridden often requiring hospitalization.

The technical factors used in the roentgen therapy of patients who manifested symptoms of irradiation sickness were similar in all cases. The kilovoltage was 210 kv. (peak). The area varied in size from 125 to 400 sq. cm. The skin-target distance was 20 inches. Filter factors varied from 0.5 mm. copper to a Thoraeus 2 filter (equivalent to 2 mm. Cu). Daily treatments were given five or six times a week. The treatment dose varied from 200 to 430 roentgens measured in air at skin distance. The maximum number of treatments per series was thirty-six and the largest series dose was 7,600 r. The onset of symptoms varied from the first to the thirteenth treatment.

The therapeutic effects of the administration of amphetamine or d-desoxyephedrine was designated as "good" if a definite remission of symptoms immediately followed. These patients were able to resume activities essentially the same as had been possible prior to the beginning of roentgen therapy. The therapeutic response was designated as "fair" when there was little improvement in the symptoms of irradiation sickness whereas the severity did not progress to cause interruption of irradiation. The patients who had a "negligible" response were those who developed symptoms of unusual severity requiring interruption or termination of roentgen therapy despite the use of either drug. The therapeutic effect of amphetamine or dextro-desoxyephedrine on 69 patients with symptoms of radiation sickness is summarized in Table I.

TABLE I

THERAPEUTIC EFFECT OF AMPHETAMINE AND D-DESOXYEPHEDRINE ON THE SYMPTOMS OF IRRADIATION SICKNESS

	Response			
	Good	Fair	Negligible	Total
Amphetamine	21	5	1	27
d-Desoxyephedrine	34	5	3	42
Total	55 (80%)	10 (14%)	4 (6%)	69

Several patients illustrate the favorable therapeutic response of the majority. One patient received prophylactic roentgen therapy after an orchidectomy revealed a seminoma. He received thirty-six treatments, each 200 r, within forty-four days, for a total of 7,200 r. Six areas were treated, each 20 by 20 cm., covering the anterior and posterior pelvis, upper abdomen, and thorax. Severe symptoms by the thirteenth treatment included marked weakness and repeated vomiting. Following medication with d-desoxyephedrine the patient resumed difficult physical labor throughout the last twenty treatments of the series. Another patient receiving roentgen therapy after a radical mastectomy had severe symptoms of roentgen sickness and was limited to 300 r applied to the thorax on alternate days. Following the administration of amphetamine the patient was able to tolerate two treatments daily, or a total of 600 r applied to the thorax each day. One patient with a lymphatic leukemia and another with a myelogenous leukemia had prostrating symptoms of irradiation

sickness which were well controlled with d-desoxyephedrine.

Ten of the 69 patients had no apparent improvement of the symptoms of irradiation sickness after administration of amphetamine or d-desoxyephedrine. They were, however, able to continue roentgen therapy without interruption. Six of these patients had extensive metastases. Only 1 of the 10 patients had an erythrocyte count above four million. One patient had severe menopausal symptoms.

All 4 patients with a "negligible" response to the administration of amphetamine or d-desoxyephedrine had extensive metastases and 3 were in a terminal state. The other had symptoms of gastrointestinal obstruction at the beginning of roentgen therapy.

The dose and method of administration of either drug are of utmost importance and must be varied according to the requirements of the individual patient. The first dose is given thirty minutes before breakfast during which time the patient remains in bed; the second dose is given at noon and the third at 4 P.M. Five to 10 mg. of amphetamine or 2.5 to 5 mg. of d-desoxyephedrine is given per dose, depending upon the size of the patient and the severity of the symptoms. The initial dose is frequently larger than the other two. The usual method is to begin with smaller doses and increase until the therapeutic effect is obtained. The total daily dose does not exceed 30 mg. of amphetamine or 20 mg. of d-desoxyephedrine. Both are administered orally. No serious side effects were noticed. An occasional patient required a barbiturate to obtain adequate rest at night. The use of either amphetamine or d-desoxyephedrine was terminated within three days after the last roentgen treatment. No evidence of habit formation was found.

MECHANISM OF ROENTGEN SICKNESS

The present study concerning the favorable therapeutic use of amphetamine and dextro-desoxyephedrine demonstrates the need for a hypothesis which explains the mechanism of irradiation sickness and the beneficial effect of diverse therapeutic methods and agents. The proposed hypothesis is suggested by recent concepts of incipient forms of shock in which the basic mechanism is an increased capillary permeability caused by various traumatic agents. Irradiation sickness, however, is not identical with the usual forms of shock because of several factors: first, there is no external blood loss; second, the trauma is relatively minor but repeated over a protracted interval of time in contrast to common forms of rapidly developing shock caused by violent trauma. Lesser degrees of trauma repeatedly applied over a longer time permit modification of typical shock by compensating mechanisms of the body. Irradiation sickness, therefore, is similar to incipient shock modified by the fact that changes occur over a period of days or weeks instead of minutes or hours. The basic mechanism of irradiation sickness will be designated as an increased capillary permeability. The nondescriptive term "shock" is confusing and designates a variety of clinical syndromes. The mechanism of radiation sickness is diagrammatically presented in Figure 1.

Roentgen rays may increase capillary permeability in two ways. One is the direct effect on capillaries within the direct path of the roentgen rays. The other is the destructive effect on pathological cells liberating certain products of cell breakdown which have a foreign protein or histamine-like reaction and cause a generalized increase in capillary permeability. This histamine-like reaction is probably present only when there are pathological cells or when the exposure of normal cells to roentgen rays is of sufficient intensity to have a lethal effect. The direct effect of roentgen rays on normal capillaries apparently is present whenever living tissue is irradiated. Pendergrass and Hodes[2] refer to experiments by Hodes and Griffith which demonstrated a marked dilatation and hyperemia in living tissues appearing soon after irradiation. Rigdon and Curl[3] discuss the mechanism and present experimental evidence of increased capillary permeability following irradiation. The direct

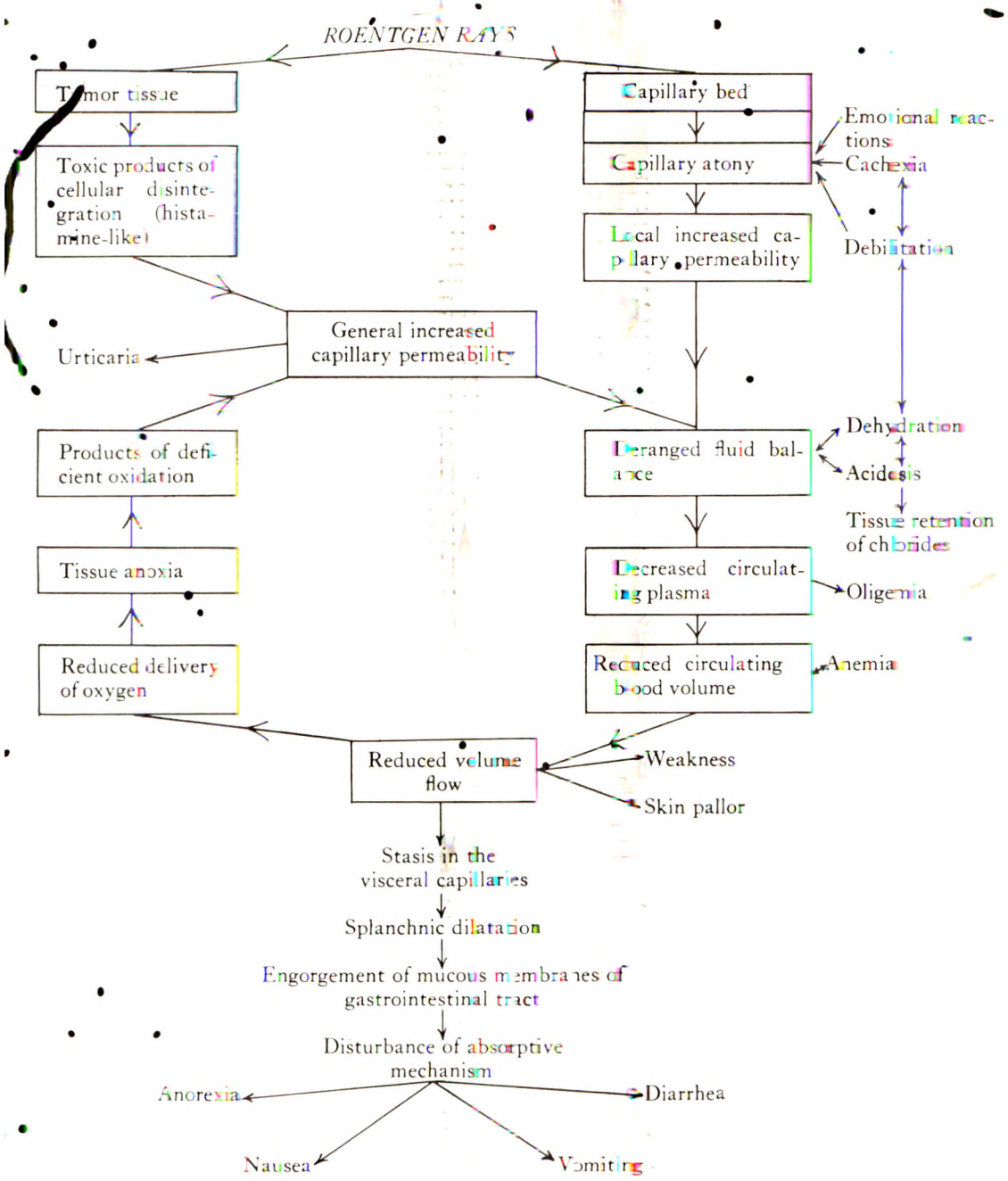


FIG. 1. Diagram showing mechanism of irradiation sickness.

effect of irradiation results in capillary permeability only in those tissues in the path of the roentgen rays. The toxic products of cellular disintegration probably affect all capillaries regardless of whether or not they are in the path of the roentgen rays.

The mechanism of the development of irradiation sickness is initiated by the increased permeability of the capillaries and a consequent diffusion of plasma fluids into the tissue spaces so that there is a disturbance of the intracellular and extracellular equilibrium of the electrolytes and fluids. This deranged fluid balance is followed by local congestion, edema, and stasis of circulating blood. A vicious cycle is intro-

duced by the subsequent reduced blood volume, decreased blood flow, insufficient delivery of oxygen, tissue anoxia, and the products of deficient oxidation which increase the damage to the capillary walls and perpetuate the condition. Of these changes, the local oligemia, injury to the capillary endothelium, and anoxia are of basic importance. Any factor which promotes these changes perpetuates the cycle. The products of deficient oxidation which are released from tissues localized in the direct path of the roentgen rays may thus cause a generalized increased capillary permeability similar to the effect of the toxic products of cellular disintegration.

The initiation and perpetuation of this cyclic mechanism of irradiation sickness is modified by many variable factors. These can be divided into two groups: the first includes the factors which modify the degree of insult and the second, factors which influence the individual's susceptibility.

The following factors influence the degree of insult:

(*a*) The number of capillaries per volume of tissue in the irradiated region has a direct influence. The epigastric region or splanchnic reservoir is the largest capillary bed in the body and irradiation in this region should cause the most severe irradiation sickness. Capillary beds in the mediastinum and lung fields are next in order of size, whereas the lower abdomen probably ranks third. This relative size of the capillary beds of the three regions mentioned coincides with the relative severity of symptoms following irradiation of these regions. The capillary beds of the extremities are limited in comparison and are well supported by the muscles, the actions of which tend to prevent dilatation and stasis of the capillaries. Irradiation of extremities seldom causes irradiation sickness except in the presence of a large tumor.

(*b*) The number of capillaries lying in the path of the roentgen rays in any given region is directly affected by the size of the field. This is controlled by the size of the cone or shield used. Larger fields cause more severe symptoms of irradiation sickness.

(*c*) The depth dose has a direct effect on increased capillary permeability, whereas the skin dose has no relationship to the quantity of radiation delivered to the large capillary beds within the trunk portion of the body.

(*d*) The time interval between treatments influences the degree of recovery of the irradiated capillaries and allows certain compensatory mechanisms to function.

The following factors influence the individual susceptibility:

(*a*) Nervous and emotional characteristics of different individuals influence their susceptibility to irradiation sickness. Increased capillary permeability occurs in people with an unstable nervous mechanism as manifested by instances of urticaria or fainting following fright or minor trauma.

(*b*) The presence and character of pathologic cells subjected to irradiation has considerable effect on irradiation sickness. Radiosensitive tumors have a more massive liberation of cellular breakdown products and more severe symptoms result.

(*c*) Anemia is a predisposing factor, since any decrease in the number of circulating erythrocytes reduces the oxygen-carrying capacity of the blood.

(*d*) Debilitation, cachexia, and dehydration predispose to irradiation sickness by their alteration in fluid balance, blood chemistry and general body resistance.

(*e*) Capillary fibrosis resulting from previous roentgen therapy probably alters the degree of increased capillary permeability caused by future irradiation.

(*f*) Changes in the suprarenal glands may be responsible for differences in susceptibility to irradiation sickness. Under normal conditions, the suprarenal gland is thought to produce sufficient cortin or a similar substance to maintain cellular permeability. Under stimulation by an insult, it is necessary to secrete an excessive amount of this protective substance. This is thought to exhaust the cortical lipids which return in about twenty-four hours and which are then followed by enlargement of the supra-

renal cortex and increased vacuolation. Frequently repeated mild insults may thus cause a suprarenal hyperplasia resulting in progressively increased resistance. However, long-continued insults, even of a mild nature, may lead to suprarenal exhaustion and atrophy with the result that the individual is extremely susceptible to many minor insults, especially to histamine-like substances.

CORRELATION OF SYMPTOMS WITH THE MECHANISM OF IRRADIATION SICKNESS

The symptoms of irradiation sickness can be correlated with the hypothesis of increased capillary permeability as the basic mechanism. Increased permeability of the capillaries of the skin and subcutaneous tissues can account for the occasional urticaria or purpura. The variable derangement of the fluid balance explains changes of varying degrees in the blood chemistry, the chloride retention of the tissues, occasional acidosis and dehydration. Changes in the fluid balance are aggravated by vomiting and diarrhea. It may be possible for irradiation to produce an apparent anemia on the basis of stasis in the visceral capillaries and splanchnic reservoir followed by a compensatory dilatation in the peripheral circulation. Muscular weakness and occasional skin pallor are readily explained by the generalized reduction in peripheral circulation causing a loss of muscle tone.

Some of the more serious symptoms of irradiation sickness can be explained on the basis of reduced blood flow which causes a stasis in the visceral capillaries and a splanchnic dilatation. This is accompanied by engorgement of the mucous membrane of the gastrointestinal tract and related organs causing a profound disturbance of the entire digestive and absorptive mechanism with symptoms of anorexia, nausea, vomiting, and diarrhea. A mucositis caused by direct irradiation of the small and large intestines contributes to the gastrointestinal symptoms.

CORRELATION OF PREVIOUS THERAPEUTIC MEASURES WITH THE MECHANISM OF ROENTGEN SICKNESS

Inconstant success of many therapeutic measures for treatment of irradiation sickness has been reported in the medical literature. The formation of ozone was once thought to cause irradiation sickness. The treatment consisted of the application of a facial mask to exclude the ozone while respiration was maintained by inhalation of oxygen. This form of therapy appears to have been of transient benefit to anoxemia. Adrenalin and ephedrine have been used and probably were beneficial in temporarily combating splanchnic dilatation. Their value is limited by the short duration of action and the ill effects of peripheral arteriolar vasoconstriction. The use of intravenous fluids has been of limited value in augmenting the plasma volume and replacing circulating electrolytes. Sedatives have been useful in some patients to reduce the psychic element and the instability of the nervous system. Liver extracts have probably had their greatest value in the presence of an anemia. Other supportive measures combat debilitation, cachexia, and dehydration. Vitamin B preparations are thought to aid in the metabolism of the products of cell disintegration. Changes in the small intestine in so-called deficiency states indicate that vitamin B preparations may influence the digestive mechanism. The use of the fractional dose method of combating irradiation sickness is successful insofar as the time interval allows compensatory mechanism to function. The recent report[6] of the beneficial effect of desoxycorticosterone acetate in the management of irradiation sickness may be due to the direct influence of this synthetic hormone on capillary permeability.

DISCUSSION OF THERAPEUTIC EFFECTS OF AMPHETAMINE AND D-DESOXYEPHEDRINE

Observation of the value of amphetamine and d-desoxyephedrine in the treatment of irradiation sickness preceded the

development of the hypothesis concerning the basic mechanism. Therefore, detailed information from hematocrit readings, capillary permeability tests, blood chemistry values, and other pertinent data which might give further support to the hypothesis, was not obtained at the time of irradiation. However, a retrospective study of the patients reveals several pertinent observations.

The value of amphetamine in irradiation sickness is suggested by some of its pharmacological actions. A protocol[4] including many reports on all aspects of the drug is the basis for evaluation of the properties of amphetamine. Consideration of its properties is limited to responses obtained by doses within the therapeutic range as used in this study. Animal experiments have demonstrated the value of the drug in various types of vasomotor collapse. A marked increase in venous tone has been found. There is no appreciable peripheral vasoconstriction such as that caused by epinephrine. The consensus of the medical literature is that oral doses of less than 2 mg. of amphetamine have little effect on blood pressure in humans unless the pressure is previously lowered. The leukocyte and erythocyte counts are considerably increased by administration of amphetamine to dogs and rabbits. This effect is due to splanchnic constriction, since it does not occur in splenectomized animals. Removal of the suprarenal glands does not diminish the response. Animal experiments reveal that contraction of the spleen begins a few minutes after injection of the drug, attains its maximum in thirty minutes, and is maintained as long as six hours. A reduction in spleen size averaging 32 per cent one hour after subcutaneous administration of 15 mg. has been observed in normal humans. The blood count is often increased, apparently due to mobilization of blood cells from the splanchnic region. The effect is variable, the leukocytes are affected more than the erythrocytes. Amphetamine has a diuretic effect on the kidneys, in contrast to epinephrine and ephedrine. A direct effect on capillary and cellular permeability has not been demonstrated. Amphetamine tends to decrease oxygen consumption and inhibits the depressor effect of experimental anoxia. The effect on the gastrointestinal tract is variable, although a generalized relaxation and inhibition of movement, especially in the lower segments, has been observed. Oral doses of 20 to 30 mg. have been shown to relax the colon. The action of amphetamine is prolonged and traces of the drug may be found after twenty-four to forty-eight hours. However, the duration of its therapeutic action is seldom more than six hours.

Dextro-desoxyephedrine acts similar to ephedrine and amphetamine. The potency per milligram is greater than amphetamine, the usual clinical dose being 3 to 9 mg. The clinically effective dose of d-desoxyephedrine has a ration of about 3 to 5 when compared to amphetamine. A report by Glomme[1] concerning the use of d-desoxyephedrine, known as pervitin, in the treatment of irradiation sickness, has been found recently in the foreign medical literature. He reported a beneficial effect of pervitin and believes that the action is due to an increased sympathicotonia.

Evaluation of the therapeutic effect of amphetamine and d-desoxyephedrine indicates that the greatest value is in combating the weakness, exhaustion, and lassitude of irradiation sickness. The mental depression frequently associated with malignant disease is usually improved. Symptoms of nausea and vomiting are nearly always improved although they may not be eliminated. Anorexia usually persists. The occasional diarrhea associated with low abdominal and pelvic irradiation is sometimes improved but often requires additional medication. Patients with a severe anemia respond less favorably. Patients having extensive metastases causing considerable interference in the physiological functions of the vital organs seldom receive benefit from the medication. The principal effect of amphetamine and d-desoxyephedrine is most likely a combating of dilatation of the

visceral capillaries and stasis in the splanchnic reservoir. A more adequate circulating blood volume is thereby maintained.

CONCLUSION

1. The therapeutic value of amphetamine and dextro-desoxyephedrine in the treatment of symptoms of irradiation sickness is demonstrated in a study of 69 patients.

2. A proposed hypothesis concerning the basic mechanism of irradiation sickness indicates the rôle of local and general increased capillary permeability. Various predisposing and modifying factors are discussed. The symptoms and previous therapeutic measures are correlated with the cyclic mechanism of increased capillary permeability.

3. The pharmacology of amphetamine and d-desoxyephedrine indicates that they are of therapeutic value in irradiation sickness by their action in maintaining the peripheral circulating blood volume and preventing visceral stasis and splanchnic dilatation. The effectiveness is reduced by anemia, nutritional and metabolic changes associated with debilitation, and by extensive metastases.

4. The present study indicates that the management of irradiation sickness should include:

(*a*) A critical evaluation of each patient's physical condition prior to roentgen therapy. Corrective measures should be instituted in the presence of anemia, dehydration, acidosis, the deficiencies associated with debilitation and nutritional disturbances.

(*b*) A prophylactic use of small oral doses of amphetamine or d-desoxyephedrine, increasing the dose with the first evidence of increasing weakness or lassitude.

(*c*) A prophylactic use of the water-soluble vitamins which are readily depleted by any disturbance of the digestive and absorptive mechanism.

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VARIATIONS IN SENSITIVENESS OF DIFFERENT SKIN AREAS TO THE ERYTHEMA DOSE OF ROENTGEN RADIATION

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IT HAS long been known that all regions of the skin surface are not equally susceptible to the erythema dose of roentgen radiation. This variable sensitivity has been commented on by several authors (Erskine, Quimby, Belisario), but there has been little systematic comparison of the sensitiveness of various skin areas by methods accurate and experimental. Because the skin in almost all parts of the body is subject at some time or other to the influence of radiation therapy directed against the ubiquitously distributed epitheliomas in the cutis itself or through the skin toward some deeper organs, it is a matter of considerable practical importance to appreciate these values in radiosensitivity.

PROCEDURE

The response of various arbitrarily selected sites in the skin to an erythema dose of roentgen rays was selected for comparative study. Twenty-one patients were used in these determinations, on whom an aggregate of 120 tests was made. The following areas of skin were chosen:

(a) Corpus sterni at the level of the nipple.
(b) Mammary line just below the areola.
(c) Axilla.
(d) Spine of sixth dorsal vertebra.
(e) Volar surface of forearm at junction of middle and lower thirds.
(f) Dorsal surface of forearm of corresponding area.
(g) Groin.
(h) Volar surface of thigh at junction of middle and lower thirds.
(i) Dorsal surface of thigh of corresponding area.
(j) Center of palm of hand.
(k) Center of dorsum of hand.
(l) Center of instep of foot.
(m) Center of dorsum of foot.

The radiation factors were 100 kv., 6 ma., 1 mm. Al, 15 cm. target skin distance, 2 cm. diameter cone, 400 r, measured in air, delivered at 234 r per minute. On any one patient, six of the above areas were exposed to the radiation stated above, which was chosen as just above the threshold erythema dose for this quality of roentgen radiation. For two to four weeks thereafter, the areas were examined daily or every other day, at approximately the same time of day, with as nearly as possible the same intensity of daylight. The observed reactions were graded from 1 to 4, a grade of 1 indicating that the complete circular area was visible but extremely faint, and 4 indicating the sharpest reaction observed, a strong erythema with subsequent scaling.

The patients ranged in age from seventeen to seventy-eight years. There were 14 males and 7 females. Nine of the patients had carcinoma of the stomach and 6 had gastric ulcer; of the remainder, 4 had malignant tumors and 2 benign growths.

RESULTS

The results are presented in Table 1, the readings being those taken on the fourteenth day after irradiation. This time was chosen because the erythema is then usually at its maximum. It is readily seen that there are variations in the erythema reaction of different parts of the body and of homologous areas in different individuals. Figure 1 constructed from Table 1 depicts the regional variations. The axilla and groin seem to be most sensitive to the radiation used; the hand and foot are least sensitive. The forearm is more sensitive than the thigh, whereas the areas over the sternum and sixth dorsal spine occupy a position intermediate between the forearm and

TABLE I

REGIONAL VARIATION IN SENSITIVENESS TO ERYTHEMA DOSE OF ROENTGEN RADIATION IN THE SAME AND DIFFERENT INDIVIDUALS

Patient	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21
Sex	M	M	F	M	M	F	M	F	M	M	M	M	F	F	M	F	M	F	M	M	M
Age	56	65	25	37	52	66	37	60	43	50	7	53	44	49	78	40	54	62	46	36	59
Complexion*	D	L	D	D	D	L	D	D	D	L	D	D	L	L	L	D	L	L	D	D	D
Sternum	1	3	3	2	3	1	2	1	3	1	3	3	1	2	0	2		0			1
Mammary line	2	3	3	2			2		2		2	2	1								
Axilla					3	2	2	0	3	3	4	4	3	3							
Sixth dorsal spine						1	2						3	3		2	1	0	1	2	1
Forearm, volar	3	3	2	2	2	1		1	2	1	3	3	2		1						
Forearm, dorsal	3	1	2	3	2	1															
Groin					3	2	2	0	3	0	4				2						
Thigh, volar	2	1	1	1			1	0	0	0	2	2	1	1	0						
Thigh, dorsal	2	1	1	1																	
Hand, palmar																1	0	0	0	0	0
Hand, dorsal																2	0	0	0	0	0
Foot, plantar														0	0	0	0	0	0	0	0
Foot, dorsal														1	0	1	0	0	1	0	0

* D = dark complexion, L = light complexion.

thigh. There is essentially no difference between the areas over the sternum and sixth dorsal spine. The sensitiveness of the dorsal and volar aspects of the forearm is approximately the same, while the dorsum of the thigh is somewhat more sensitive than its volar aspect. The axilla is more sensitive than the groin. There is apparently no difference between the dorsum of the hand and that of the foot. The palm of the hand exhibited slight sensitiveness, a minimal reaction in 1 out of 6 individuals tested, whereas there was no reaction elicited on the plantar surface of the foot in 8 persons investigated.

In most instances, it was found that all the areas tested on any one subject develop an erythema at about the same time, with the exception of the axilla and groin. These areas frequently require twenty-four to seventy-two hours longer. Table II illustrates this point in a representative case.

There does not appear to be any appreciable variation in the intensity of the reaction on the basis of sex or complexion in these patients, all but one of whom were Caucasian, the exception being a Korean. The experiment has not been attempted on the black, yellow, brown or red races. As a matter of interest, a low sensitiveness was shown by Patient 10 who was red-haired. Some of the subjects were elderly patients who were debilitated from a chronic illness so that their skin was thin and atrophic. The test areas on these persons were somewhat less sensitive than the homologous ones in younger, more robust individuals.

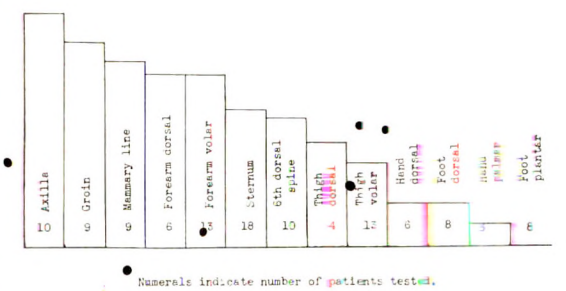


FIG. 1. Comparative sensitiveness of various skin areas to erythema dose of low voltage roentgen rays.

DISCUSSION

The lowered sensitiveness of the hand and foot may be of practical value in radiation therapy. Because the skin on these extremities is apparently less sensitive than elsewhere, it might be reasoned that tumors requiring radiation therapy would need correspondingly larger doses. For the same reason, neoplastic lesions of the thigh might require and initially tolerate more intensive irradiation than similar tumors of the forearm, whereas those of the axilla and groin should be treated more circumspectly.

A distinction must be made between the initial susceptibility of the skin as evidenced by the time of appearance and degree of erythema and the late or eventual tolerance of the same skin to a complete therapeutic roentgen-ray dose. The two responses are not comparable, because skin that is relatively ischemic, such as over the vertebral spine, may show little initial response to an erythema dose but may undergo late radiation necrosis following a dose of roentgen rays or radium that the relatively more susceptible axillary skin would tolerate with safety. The same may be said for the sole of the foot, the skin of which is relatively insensitive to irradiation, as these tests indicate, yet may break down and ulcerate after moderate therapeutic irradiation, due perhaps to the inability of such irradiated skin to tolerate the pressure of weight bearing to which the sole is subjected. These facts only serve to emphasize the statements previously made, that our experiments reported herein are based solely on the initial response of various skin areas to an erythema dose of low voltage roentgen rays.

Our results demonstrate that there is a variation in the sensitiveness of diverse skin areas of the body to low voltage roentgen rays. The reason for such differences is not clear although several theories have been advanced. Forak, working with high voltage roentgen rays, notes that areas of skin lying over or in close proximity to bone exhibit a decreased sensitiveness to irradiation. This might explain our finding of markedly diminished sensitiveness over the hand and foot, were it not for the fact that the areas over the sternum and spine of the sixth dorsal vertebra are relatively sensitive.

Desjardins believes that the circulation plays an important rôle. Those tissues which are ischemic have a lessened degree of irradiation reaction as compared with those possessed of normal circulation. This cannot be used to explain the diminished sensitiveness of the hands of our subjects, although it might account for that of the feet, since some of those tested were elderly and somewhat arteriosclerotic. By referring to Table I it may be seen that those persons who developed an erythema on the dorsum of the foot were under fifty years of age.

Another explanation is suggested by Belisario. He quotes the work of Maximow and Bloom to show that the skin in differ-

TABLE II

VARIATIONS IN LATENT PERIOD INTERVENING BETWEEN TIME OF IRRADIATION AND APPEARANCE OF ERYTHEMA IN THE SAME INDIVIDUAL (PATIENT 6)

Days after irradiation	1	2	3	4	5	7	16	18	21	25
Area tested:										
Corpus sterni	1	1	1	1	1	1	1	1	2	2
Groin	0	0	1	1	1	2	2	3	3	3
Axilla	0	0	0	0	1	1	2	3	3	3
Sixth dorsal spine	0	1	1	1	1	1	2	3	3	3
Forearm, dorsal	0	1	1	1	1	1	1	2	2	2
Forearm, volar	0	1	1	1	1	1	1	1	2	2

ent parts of the body varies in thickness, the palm and sole being of greatest thickness. He believes that these differences in skin thickness explain the variations in sensitiveness to irradiation. This may be a factor but it fails to account for the diminished sensitiveness of the relatively thin skin of the dorsum of the hand and foot.

It is probable that circulation and thickness of skin play a rôle in the phenomenon of sensitiveness. However, this is not the entire explanation. The axilla and groin, where the most intense reaction is obtained, contain many oil and sweat glands and the skin in these locations is thinner, more moist and usually of higher temperature than elsewhere.

There is a diversity of opinion as to whether the threshold erythema of blonds and brunets differs. Some investigators (Erskine, Nisbet and Keatinge, Goldsmith) believe that blonds are more sensitive, while others (Laymon and Cumming, Belisario) hold to the contrary. Our investigations indicate that there is no appreciable difference in sensitiveness.

SUMMARY

1. The sensitiveness of thirteen different skin areas to low voltage roentgen rays was tested in a group of twenty-one patients.

2. There is a variation in sensitiveness of different skin areas in the same person and of homologous areas in different persons.

3. The axilla and groin are most sensitive, whereas the hand and foot are the least sensitive of the areas tested. The axilla is more sensitive than the groin, and the dorsum of the hand and foot more sensitive than the palm or sole. Between these extremes lie the other areas in the following order of decreasing sensitiveness: mammary line just below the areola, forearm (dorsal and volar), sternum, sixth dorsal spine, thigh (dorsal and volar).

4. In most instances all the areas tested on any one subject develop an erythema at about the same time, with the exception of the axilla and groin which frequently require twenty-four to seventy-two hours longer.

5. There does not appear to be any appreciable difference in sensitiveness on the basis of sex or complexion. The skin of elderly, debilitated patients, however, appears less sensitive than that of younger, more robust individuals.

6. The variations in sensitiveness may be of practical importance in the treatment of lesions on various parts of the body.

7. Theories advanced to explain differences in sensitiveness are briefly examined in the light of the present investigation.

The authors wish to acknowledge their indebtedness to Dr. Edith H. Quimby for her supervision of the details of the irradiation method and for her suggestions and assistance during the course of the work.

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EDITORIALS

WELDERS' LUNGS

IN THE November, 1942, issue of this JOURNAL there appeared an editorial[1] on "Metal Fume Fever and Nitric Fume Pneumonia," two clinical entities sometimes encountered in electric arc welders. In that editorial it was stated that, as far as could be ascertained, no definite late roentgen changes occurred in welders' lungs, although there were some scattered reports of such in the literature. Since writing that editorial we have encountered one case of pulmonary nodulation in a welder who had never been exposed to significant amounts of silica, and we have been apprised of the further studies of Enzer and Sander on this problem.[2,3]

These observers have examined a large number of electric arc welders who used bare metal electrodes for a number of years. They found variable degrees of diffuse nodulation in the chest roentgenograms of some workers (usually men who had worked for seven or more years, and had been exposed to exceptionally massive concentrations of welding fumes, for example, when welding the inside of steel tanks, etc.). They secured autopsy studies on cases dying of incidental causes and ascertained the fact that small deposits of iron oxide were present throughout the lungs, in the perivascular spaces and lymphatics. There was no associated fibrosis.

The roentgenogram of our case, and of the cases seen by Enzer and Sander, showed diffuse pulmonary nodulation, the densities varying from 1 to 3 mm. in diameter and closely resembling the densities seen in some cases of silicosis. There was not the hilar adenopathy or hilar density often seen in silicosis. However, at first glance, the roentgen appearance was strikingly similar to that of silicotic nodulation.

It is stressed (in the autopsy reports) that iron oxide dust is the only one definitely identified so far. The term siderosis could therefore be applied to the condition, but might cause confusion with the siderosis hitherto verified best at autopsy. Most cases of nodulation occurring in iron workers and labeled siderosis have been shown to be cases of fibrosis secondary to coincidental exposure to silica.

The absence of fibrosis in cases of welders' nodulation is noteworthy, and is borne out by the fact that clinically these men showed no functional impairment of the lungs, no tendency to complicating infection, and no clinical symptoms. The nodulation was found on routine roentgen examinations incidental to employment.

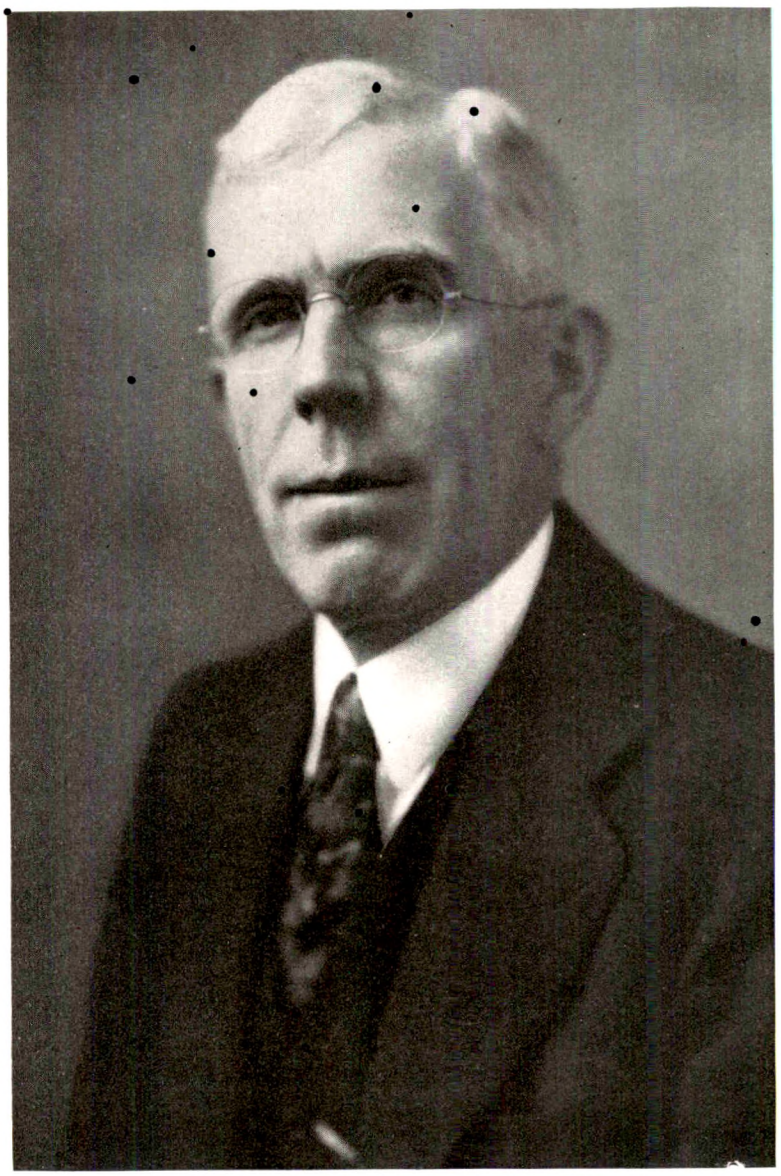
In summary, therefore, it is apparent that, contrary to previous opinion, some electric arc welders, exposed to unusually heavy concentrations of fumes over long periods of time (perhaps seven or more years), will develop benign pulmonary nodulation, due to the deposition of iron oxide in the lungs and not to fibrosis.

L. HENRY GARLAND

[1] Garland, L. H. Metal fume fever and nitric fume pneumonia (editorial). AM. J. ROENTGENOL. & RAD. THERAPY, 1942, *47*, 692–693.

[2] To be published.

[3] Enzer, N., and Sander, O. A. Chronic lung changes in electric arc welders. *J. Indust. Hyg. & Toxicol.*, 1938, *20*, 333–350.



DAVID YANDELL KEITH
1881-1943

DR. DAVID YANDELL KEITH of Louisville, Kentucky, died on July 12, 1943, of heart disease.

Dr. Keith was born in Crofton, Kentucky, April 8, 1881, and in his youth worked variously as a tobacco factory hand, school teacher, clerk, insurance salesman and druggist's assistant. In 1909 he was graduated as honor man from the University of Louisville Medical Department. Filling in the summer vacation that year as sanitary inspector for the city of Hopkinsville, Kentucky, he began his internship in September to serve for a year in the Louis-

ville City Hospital. He served on the faculty of the University of Louisville School of Medicine almost from the time of his graduation. His occupation with radiology began in 1912, when he installed a roentgen-ray machine in his office in partnership with Dr. G. S. Hanes. Later partners or assistants were his brother, Dr. J. Paul Keith, Dr. H. C. Herrmann, Dr. J. C. Bell, Dr. E. L. Shiflett, and Dr. H. L. Townsend. The latter assumed Dr. Keith's practice and is continuing his work at the Kentucky Baptist Hospital.

From 1914 to 1931, Dr. Keith was radiologist to the St. Mary Elizabeth Hospital and he served similarly the Kentucky Baptist Hospital from its organization in 1924. In 1920 he became radium therapist to the Louisville City Hospital. He was consulting radiologist to the Marine Hospital from 1913 on. He was also on the staff of the Children's Free Hospital and the Deaconess Methodist Hospital. His membership in the American Roentgen Ray Society dates from 1916. He was also a member of the American Radium Society, American Medical Association, Kentucky State and Jefferson County Medical Societies, local Society of Physicians and Surgeons, and a Fellow of the American College of Radiology.

Dr. Keith is survived by his wife, who was Miss Vonda Barth, to whom he was married in 1916, by a daughter, Vonda Barth Keith, and a son, David Yandell Keith, Jr, a private first class in the Army and a senior student at the University of Louisville Medical School.

RAMSAY SPILLMAN

SOCIETY PROCEEDINGS, CORRESPONDENCE AND NEWS ITEMS

Items for this section solicited promptly after the events to which they refer.

MEETINGS OF ROENTGEN SOCIETIES*

UNITED STATES OF AMERICA

AMERICAN ROENTGEN RAY SOCIETY
Secretary, Dr. H. Dabney Kerr, University Hospital, Iowa City, Iowa. Annual meeting: Joint Meeting of American Roentgen Ray Society and Radiological Society of North America, Palmer House, Chicago, Ill., Sept. 24–29, 1944.

AMERICAN COLLEGE OF RADIOLOGY
Secretary, Mac F. Cahal, 540 N. Michigan Ave., Chicago, Ill.

SECTION ON RADIOLOGY, AMERICAN MEDICAL ASSOCIATION
Secretary, Dr. J. T. Murphy, 421 Michigan St., Toledo, Ohio. Annual meeting: Chicago, Ill., June 12–16, 1944.

ARKANSAS RADIOLOGICAL SOCIETY
Secretary, Dr. J. S. Wilson, Mack Wilson Hospital, Monticello, Ark. Meets every three months and also at time and place of State Medical Association.

RADIOLOGICAL SOCIETY OF NORTH AMERICA
Secretary, Dr. D. S. Childs, 607 Medical Arts Bldg., Syracuse, N. Y. Annual meeting: Joint Meeting of American Roentgen Ray Society and Radiological Society of North America, Palmer House, Chicago, Ill., Sept. 24–29, 1944.

RADIOLOGICAL SECTION, BALTIMORE MEDICAL SOCIETY
Secretary, Dr. Walter L. Kilby, Baltimore. Meets third Tuesday each month, September to May.

SECTION ON RADIOLOGY, CALIFORNIA MEDICAL ASSOCIATION
Secretary, Dr. Earl R. Miller, University of California Hospital, San Francisco, Calif.

RADIOLOGICAL SECTION, CONNECTICUT MEDICAL SOCIETY
Secretary, Dr. Max Climan, 242 Trumbull St., Hartford, Conn. Meets bi-monthly on second Thursday, at place selected by Secretary. Annual meeting in May.

SECTION ON RADIOLOGY, ILLINOIS STATE MEDICAL SOCIETY
Secretary, Dr. H. W. Ackemann, 321 W. State St., Rockford, Ill.

RADIOLOGICAL SECTION, LOS ANGELES COUNTY MEDICAL ASSOCIATION
Secretary, Dr. Roy W. Johnson, 1407 S. Hope St., Los Angeles, Calif. Meets on second Wednesday of each month at the County Society Building.

RADIOLOGICAL SECTION, SOUTHERN MEDICAL ASSOCIATION
Secretary, Dr. Roy G. Giles, Temple, Texas.

BROOKLYN ROENTGEN RAY SOCIETY
Secretary, Dr. Leo Harrington, 880 Ocean Ave., Brooklyn, N.Y. Meets monthly on fourth Tuesday, October to April.

BUFFALO RADIOLOGICAL SOCIETY
Secretary, Dr. Joseph S. Gian-Franchèschi, 610 Niagara St., Buffalo, N. Y. Meets second Monday of each month except during summer months.

CHICAGO ROENTGEN SOCIETY
Secretary, Dr. Warren W. Furey, 6844 S. Oglesby Ave., Chicago, Ill. Meets second Thursday of each month October to April inclusive at the Palmer House.

CINCINNATI RADIOLOGICAL SOCIETY
Secretary, Dr. Samuel Brown, 707 Race St., Cincinnati, Ohio. Meets third Tuesday of each month, October to May, inclusive.

CLEVELAND RADIOLOGICAL SOCIETY
Secretary, Dr. D. D. Brannan, 11311 Shaker Blvd., Cleveland 4, Ohio. Meets at 6:30 P.M. at Allerton Hotel on fourth Monday each month, October to April, inclusive.

DALLAS-FORT WORTH ROENTGEN STUDY CLUB
Secretary, Dr. X. R. Hyde, Medical Arts Bldg., Fort Worth, Texas. Meetings held in Dallas on odd months and in Fort Worth on even months, on third Monday, at 7:30 P.M.

DENVER RADIOLOGICAL CLUB
Secretary, Dr. Edward J. Meister, 366 Metropolitan Bldg., Denver, Colo. Meets third Friday of each month at Denver Athletic Club.

DETROIT ROENTGEN RAY AND RADIUM SOCIETY
Secretary, Dr. E. R. Witwer, Harper Hospital. Meets monthly on first Thursday from October to May, at Wayne County Medical Society Building.

FLORIDA RADIOLOGICAL SOCIETY
Acting Secretary, Dr. Walter A. Weed, 204 Exchange Bldg., Orlando, Fla. Meetings in May and November.

GEORGIA RADIOLOGICAL SOCIETY
Secretary, Dr. James J. Clark, 478 Peachtree St., Atlanta, Ga. Meets in November and at annual meeting of Medical Association of Georgia in the spring.

RADIOLOGICAL SOCIETY OF KANSAS CITY
Secretary, Dr. Arthur B. Smith, 800 Argyle Bldg., Kansas City, Mo. Meets third Thursday of each month at a place designated by the president.

ILLINOIS RADIOLOGICAL SOCIETY
Secretary, Dr. Wm. DeHollander, St. John's Hospital, Springfield, Ill. Meets three times a year.

INDIANA ROENTGEN SOCIETY
Secretary, Dr. H. C. Ocasner, Methodist Hospital, Indianapolis. Meeting held the second Sunday in May annually.

KENTUCKY RADIOLOGICAL SOCIETY
Secretary, Dr. W. C. Martin, 321 W. Broadway, Louisville. Meets annually in Louisville on first Saturday in Apr.

LONG ISLAND RADIOLOGICAL SOCIETY
Secretary, Dr. Marcus Wiener, 1430-48th St., Brooklyn, N. Y. Meets Kings County Med. Soc. Bldg. monthly on fourth Thursday, October to May, 8:30 P.M.

LOUISIANA RADIOLOGICAL SOCIETY
Secretary, Dr. J. R. Anderson, 1130 Louisiana Ave., Shreveport. Meets annually during Louisiana State Medical Society Meeting.

MICHIGAN ASSOCIATION OF ROENTGENOLOGISTS
Secretary, Dr. E. M. Shebesta, 1429 David Whitney Bldg., Detroit. Three meetings a year, Fall, Winter, Spring.

MILWAUKEE ROENTGEN RAY SOCIETY
Secretary, Dr. C. A. H. Fortier, 231 W. Wisconsin Ave., Milwaukee, Wis. Meets monthly on second Monday at University Club.

MINNESOTA RADIOLOGICAL SOCIETY
Secretary, Dr. Annette T. Stenstrom, 1218 Medical Arts Bldg., Minneapolis, Minn. One meeting a year at time of Minnesota State Medical Association.

NEBRASKA RADIOLOGICAL SOCIETY
Secretary, Dr. D. A. Dowell, Medical Arts Bldg., Omaha, Nebr. Meets third Wednesday of each month, at 6 P.M. at either Omaha or Lincoln.

NEW ENGLAND ROENTGEN RAY SOCIETY
Secretary, Dr. Hugh F. Hare, Lahey Clinic, Boston, Mass. Meets monthly on third Friday, Boston Medical Library.

RADIOLOGICAL SOCIETY OF NEW JERSEY
Secretary, Dr. H. J. Perlberg, 921 Bergen Ave., Jersey City. Meets annually at time and place of State Medical Society. Mid-year meetings at place chosen by president.

NEW YORK ROENTGEN SOCIETY
Secretary, Dr. Ramsay Spillman, 115 East 61st St., New York City. Meets monthly on third Monday, New York Academy of Medicine, at 8:00 P.M.

NORTH CAROLINA ROENTGEN RAY SOCIETY
Secretary, Dr. Major Fleming, Rocky Mount, N. C. Annual meeting at time and place of State Medical Society. Mid-year scientific meeting at place designated.

* Secretaries of Societies not here listed are requested to send the necessary information to the Editor.

NORTH DAKOTA RADIOLOGICAL SOCIETY
Secretary, Dr. L. A. Nash, St. John's Hospital, Fargo. Meetings held by announcement.

CENTRAL NEW YORK ROENTGEN RAY SOCIETY
Secretary, Dr. C. F. Potter, 820 S. Crouse Ave., Syracuse. Three meetings a year. January, May, November.

OHIO RADIOLOGICAL SOCIETY
Secretary, Dr. J. E. McCarthy, 707 Race St., Cincinnati. Meets at time and place of annual meeting of Ohio State Medical Association.

PACIFIC ROENTGEN SOCIETY
Secretary, Dr. L. H. Garland, 450 Sutter St., San Francisco, Calif. Meets annually, during meeting of California Medical Association.

PENNSYLVANIA RADIOLOGICAL SOCIETY
Secretary, Dr. L. E. Wurster, 416 Pine St., Williamsport. Annual meeting at Penn Alto Hotel, Altoona, Pa., May 13–14, 1914.

PHILADELPHIA ROENTGEN RAY SOCIETY
Secretary, Dr. R. P. Barden, University Hospital. Meetings first Thursday of each month from October to May inclusive at 8:15 P.M., in Thompson Hall, College of Physicians, 19 S. 22d St.

PITTSBURGH ROENTGEN SOCIETY
Secretary, Dr. R. G. Alley, 4800 Friendship Ave. Meets second Wednesday each month, 4:30 P.M., October to June, Pittsburgh Academy of Medicine.

ROCHESTER ROENTGEN RAY SOCIETY, ROCHESTER, N. Y.
Secretary, Dr. Sidney Larson, Strong Memorial Hospital. Meets monthly on third Monday from October to May, inclusive, 8 P.M. at Strong Memorial Hospital.

ROCKY MOUNTAIN RADIOLOGICAL SOCIETY
Secretary, Dr. A. M. Popma, 220 N. First St., Boise, Idaho.

ST. LOUIS SOCIETY OF RADIOLOGISTS
Secretary, Dr. E. W. Spinzig, 2646 Potomac, St. Louis, Mo. Meets fourth Wednesday of each month, except June, July, August, and September, at a place designated by the president.

SAN DIEGO ROENTGEN SOCIETY
Secretary, Dr. Henry L. Jaffe, Naval Hospital, Balboa Park, San Diego, Calif. Meets monthly on first Wednesday at dinner.

SAN FRANCISCO RADIOLOGICAL SOCIETY
Secretary, Dr. Martha Mottram, 450 Sutter St., San Francisco. Meets monthly on third Thursday at 7:45 P.M., first six months of year at Toland Hall, University of California Hospital, second six months at Lane Hall, Stanford University Hospital.

SHREVEPORT RADIOLOGICAL CLUB
Secretary, Dr. R. W. Cooper, Charity Hospital, Shreveport, La. Meets monthly on third Wednesday, at 7:30 P.M., September to May inclusive.

SOUTH CAROLINA X-RAY SOCIETY
Secretary, Dr. T. A. Pitts, Baptist Hospital, Columbia, S. C. Meets in Charleston on first Thursday in November, also at the time and place of South Carolina State Medical Association.

TENNESSEE RADIOLOGICAL SOCIETY
Secretary, Dr. J. M. Frère, 707 Walnut St., Chattanooga, Tenn. Meets annually at the time and place of the Tennessee State Medical Association.

TEXAS RADIOLOGICAL SOCIETY
Secretary, Dr. Herman Klapproth, Sherman, Texas.

UNIVERSITY OF MICHIGAN DEPARTMENT OF ROENTGENOLOGY STAFF MEETING
Meets each Monday evening from September to June, at 7 P.M. at University Hospital.

UNIVERSITY OF WISCONSIN RADIOLOGICAL CONFERENCE
Secretary, Dr. E. A. Pohle, 1300 University Ave., Madison, Wis. Meets every Thursday from 4:00–5:00 P.M., Room 301, Service Memorial Institute.

VIRGINIA RADIOLOGICAL SOCIETY
Secretary, Dr. E. L. Flanagan, 116 E. Franklin St., Richmond, Va. Meets annually in October.

WASHINGTON STATE RADIOLOGICAL SOCIETY
Secretary, Dr. Thomas Carlile, 1115 Terry St., Seattle. Meets fourth Monday each month, October through May, College Club, Seattle.

X-RAY STUDY CLUB OF SAN FRANCISCO
Secretary, Dr. J. M. Robinson, University of California Hospital. Meets monthly in evening on third Thursday.

CUBA

SOCIEDAD DE RADIOLOGÍA Y FISIOTERAPIA DE CUBA
President, Dr. J. Manuel Viamonte, Hospital Mercedes, Habana, Cuba. Meets monthly in Habana.

BRITISH EMPIRE

BRITISH INSTITUTE OF RADIOLOGY INCORPORATED WITH THE RÖNTGEN SOCIETY
Medical Members' meeting held monthly on third Friday at 2:30 P.M. and Ordinary Meeting at same time on following Saturday, October to May, 32 Welbeck St., London, W.1.

SECTION OF RADIOLOGY OF THE ROYAL SOCIETY OF MEDICINE (CONFINED TO MEDICAL MEMBERS
Meets on the third Friday of each month at 4:45 P.M. at the Royal Society of Medicine 1, Wimpole St., London, W. 1.

FACULTY OF RADIOLOGISTS
Secretary, Dr. M. H. Jupe, 32 Welbeck St., London, W. 1 England.

SECTION OF RADIOLOGY AND MEDICAL ELECTRICITY, AUSTRALASIAN MEDICAL CONGRESS
Secretary, Dr. H. M. Cutler, 139 Macquarie St., Sydney, New South Wales.

RADIOLOGICAL SECTION OF THE VICTORIAN BRANCH OF THE BRITISH MEDICAL ASSOCIATION
Secretary, Dr. Keith Hallam, St. George's Hospital, E.E.W., Melbourne, E. 4, Victoria, Australia. Meets monthly from March to November inclusive.

CANADIAN ASSOCIATION OF RADIOLOGISTS
Secretary, Dr. A. D. Irvine, 540 Tegler Bldg., Edmonton, Alberta.

SECTION OF RADIOLOGY, CANADIAN MEDICAL ASSOCIATION
Secretary, Dr. C. M. Jones, Inglis St., Exc. Halifax, N. S.

RADIOLOGICAL SECTION, NEW ZEALAND BRITISH MEDICAL ASSOCIATION
Secretary, Dr. Colin Anderson, Invercargill, New Zealand. Meets annually.

SOUTH AMERICA

SOCIEDAD ARGENTINA DE RADIOLOGIA
Secretary, Dr. Guido Gotta, Buenos Aires, Argentina. Meetings are held monthly.

CONTINENTAL EUROPE

SOCIEDAD ESPANOLA DE RADIOLOGIA Y ELECTRICIDAD
Secretary, Dr. J. Martin-Crespo, Fuencarral, 7, Madrid, Spain. Meets monthly in Madrid.

SOCIÉTÉ SUISSE DE RADIOLOGIE (SCHWEIZERISCHE RÖNTGEN-GESELLSCHAFT)
Secretary for French language, Dr. A. Grosjean, La Chaux de Fonds.
Secretary for German language, Dr. Schaurer, Molzgasse Biel. Meets annually in different cities.

SOCIETATEA ROMANA DE RADIOLOGIE SI ELECTROLOGIE
Secretary, Dr. Oscar Meller, Str. Banul Manăcine, 30, S. I., Bucuresti, Roumania. Meets second Monday in every month with the exception of July and August.

ALL-RUSSIAN ROENTGEN RAY ASSOCIATION, LENINGRAD
USSR in the State Institute of Roentgenology and Radiology, 6 Roentgen St.
Secretaries, Drs. S. A. Reinberg and S. G. Simonson. Meets annually.

LENINGRAD ROENTGEN RAY SOCIETY
Secretaries, Drs. S. G. Simonson and G. A. Gusterin. Meets monthly, first Monday at 8 o'clock, State Institute of Roentgenology and Radiology, Leningrad.

MOSCOW ROENTGEN RAY SOCIETY
Secretaries, Drs. L. L. Holst, A. W. Ssawygin and S. T. Konobejevsky. Meets monthly, first Monday, 8 P.M.

SCANDINAVIAN ROENTGEN SOCIETIES
The Scandinavian roentgen societies have formed a joint association called the Northern Association for Medical Radiology, meeting every second year in the different countries belonging to the Association.

APPEARANCE OF OSSIFICATION CENTERS

To the Editor:

Pyle and Sontag[1] report that "Francis and Werle calculated their tables for age of appearance from the ages when they saw the bone center for the first time on a roentgenogram. Their roentgenograms were taken at three or six month intervals. Therefore an epiphysis which began to calcify at about four months was assigned the skeletal age of six months, since that was the first roentgenogram in which it was seen. All investigators, including Flecker, have followed the same procedure." Surely I have not done so. My method is clearly stated on page 102 thus:[2]

"With regard to the various descriptions which follow, the following points with reference to each sex have been specially noted:

(a) Ages of youngest subjects in whom ossific centers in question were found to be present.

(b) Ages at which in at least half of the individual centers were found to be present.

(c) Ages of oldest subjects in whom epiphyses were found to be fused with the diaphyses.

(d) Ages of youngest subjects in whom epiphyses were found to be fused with the diaphyses.

(e) Ages at which in at least half of the individuals epiphyses were found to be fused.

(f) Ages of oldest subjects in whom epiphyses were found to be fused with the diaphyses.

The only ages given are those at which there are found to be variations. At other ages, the centers have been found to be either all present or all absent."

Admittedly the method of conducting periodical roentgenological examinations on the epiphyses of specially selected children is a great advantage over earlier procedures, provided a sufficient number of such children have been studied, but I have already condemned the prevailing practice of expressing such results in percentages. Francis and Werle[3] likewise write of 80 percentile. Presumably 4 out of 5 would be considered an 80 percentile! One might as well estimate the percentage of males and females born in the general population by noting the number of each sex born among 50 births at any institution; yet in Pyle and Sontag's work, which gives from 31 to 50 subjects in each group, they also speak of an 80th or 20th percentile. With 31 subjects in a group, it is presumed that roughly 25 or 6 would represent an 80 or 20 percentile.

The expression "coefficient of variability (per cent)" should surely not be applied to such small numbers, and only tends to discredit work on this subject. The assumption of the term percentage (percentile) implies an accuracy which cannot be attained unless considerably more than a hundred subjects are involved, and to be at all accurate many hundreds are needed.

H. Flecker

52 Abbott St.,
Cairns,
North Queensland, Australia

[1] Pyle, I., and Sontag, L. W. Variability in onset of ossification in epiphyses and short bones of the extremities. Am. J. Roentgenol. & Rad. Therapy, 1943, *49*, 795–798.

[2] Flecker, H. Time of appearance and fusion of ossification centers as observed by roentgenographic methods. Am. J. Roentgenol. & Rad. Therapy, 1942, *47*, 97–159.

[3] Francis, C. C., and Werle, P. B. Appearance of centers of ossification from birth to 5 years. *Am J. Phys. Anthrop.*, 1939, *24*, 273–299.

CANCER TEACHING DAY

A program under the title of Cancer Teaching Day and arranged under the auspices of a number of medical societies of the State of New York will be presented at Ellis Hospital, Schenectady, New York, on Thursday, April 20, 1944. The meeting will be called to order at 3:00 P M., and the following program will be presented:

Chairman, Ellis Kellert, M.D., Director, Ellis Hospital Laboratory.

Modern Trends in Cancer Research. William H. Woglom, M.D., New York, N. Y.

Tumors of the Peripheral Nerves and the Adipose Tissues. Arthur Purdy Stout, M.D., New York, N. Y.

Dinner will be served at 6:30 P.M. at the Mohawk Golf Club, after which the program will be resumed at 8 o'clock, and the following papers will be presented:

Chairman, Charles E. Rourke, M.D., President, Medical Society of the County of Schenectady.

Carcinoma of the Stomach; Results of Studies in a Surgical Clinic. The Responsibility of the General Practitioner and the Surgeon. Fordyce B. St. John, M.D., New York, N. Y.

Tumors of the Major Salivary Glands. Hayes Martin, M.D., New York, N. Y.

BOOK REVIEWS

Books sent for review are acknowledged under: Books Received. This must be regarded as a sufficient return for the courtesy of the sender. Selections will be made for review in the interest of our readers as space permits.

AN ATLAS OF ANATOMY. By. J. C. Boileau Grant, M.C., M.B., Ch.B., F.R.C.S. (Edin.), Professor of Anatomy in the University of Toronto. In two volumes. Volume I. Upper Limb, Abdomen, Perineum, Pelvis, and Lower Limb. Cloth. Price, $5.00. Pp. 214, with 227 illustrations. Baltimore, Maryland: Williams & Wilkins Company, 1943.

This work differs from other anatomies in that it presents regions, from bone to integument. It is superbly illustrated by Mrs. Dorothy I. Chubb, who was a pupil of Max Broedel, and other able artists who are identified in the preface. The regional approach makes it of especial value to roentgenologists, and anyone who has seen the first volume can scarcely do other than eagerly look forward to the second. Excellent line drawings of the bones are by Sergeant Douglas Baker, and the origins and insertions of muscles are charted in color. The letter-press is concise but adequate. The pages are 21.5×28 cm., making a convenient size of volume. Dr. Grant and his co-workers are to be congratulated on every aspect of their product, and deserve the gratitude of their colleagues for making so much anatomical information available in so attractive a form.

RAMSAY SPILLMAN

NEUROLOGY. By Roy R. Grinker, M.D., Chairman, The Department of Neuropsychiatry of the Michael Reese Hospital, Chicago. Third edition. Revised and largely rewritten. With the assistance of Norman A. Levy, M. D., Associate Neuropsychiatrist, Michael Reese Hospital, Chicago. With a chapter on Brain Tumors by Paul C. Bucy, M.D., Professor of Neurology and Neurological Surgery, University of Illinois, College of Medicine, Chicago. Cloth. Price, $6.50. Pp. 1136, with 416 illustrations. Springfield, Illinois: Charles C Thomas, 1943.

This excellent textbook of clinical neurology has now reached its third edition and is revised and rewritten and at the same time considerably enlarged. In this edition, the chapter on Brain Tumors has been written by P. C. Bucy. This chapter is well done and the subject has been effectively brought up to date. Several new techniques of neurological examination are included. Electroencephalography is described in three portions of the book—first as one of the methods of examinations and then in its application to the diagnosis of brain neoplasms and in connection with the epilepsies. Another newly added section deals with lipiodography. The reviewer believes that Grinker is probably right when he says "Cases in which iodized oil is necessary for diagnosis are rare. Its use would be greatly reduced if more detailed examination and greater thought were expended clinically before hasty reliance is placed on a mechanical procedure."

There is an especially satisfactory chapter on the neurological complications of pernicious anemia. Many other especially commendable chapters well might be listed.

To turn instead to criticisms—they are small ones—why is the word pathology used incorrectly when pathological changes are meant? While ergotism, ptomaine and mussel poisoning are discussed under the toxicities associated with food products, botulism is strangely omitted. It would seem that tetany is not too well covered. It is unfortunate that figures for blood calcium in tetany—the normal and the symptom producing—are not in the text. And incidental to items of this category, it should be possible for the reader to look up figures bearing upon the urinary output of lead or arsenic in cases of suspected poisoning with the heavy metals. True enough, there are no "normals" but the maximum amounts consistent with the preservation of health are available and should be in this type of text for reference. It is questionable whether Jamaica ginger peripheral neuropathy deserves quite all the space given to it.

On the other hand, there is not enough discussion of causalgia and the allied physiopathic nerve affections which Babinski and Froment have called reflex-paralyses. Grinker covers this field only briefly. Is his maintenance of Weir

Mitchell's term "ascending neuritis" desirable? American neurological textbooks in general pay insufficient attention to this syndrome. Probably the author's feeling that space was lacking kept him from as complete a discussion of psychogenic headache as might be wished.

But all in all, the splendid points of the book far outweigh minor deficiencies. There is no better textbook on neurology and it can be strongly recommended.

THOMAS K. DAVIS

TRANSURETHRAL PROSTATECTOMY. By Reed M. Nesbit, M.D., F.A.C.S., Associate Professor of Surgery, University of Michigan Medical School, in charge of the Section of Urology, Department of Surgery. With a Chapter on the Vascular Supply of the Prostate Gland by Rubin H. Flocks, M.D. Cloth. Price, $7.50. Pp. 192, with 62 plates including 94 original drawings by William P. Didusch. Springfield, Illinois: Charles C Thomas, 1943.

In this monograph Nesbit describes the management of prostatism by transurethral resection as conducted at his clinic at the University of Michigan Medical School. There then follows his appraisal of this operation, after which is discussed the history of this method of prostatectomy, to which is appended an extensive bibliography.

The description of the technique of enucleation of the prostate is admirably set forth by means of serial illustrations in half tone by William P. Didusch. Thus, the very difficult problem of exact demonstration of the method of operation has been met more clearly and satisfactorily than in any work heretofore published.

In evaluation of the place of the transurethral operation the writer's views appear to be very sound. Emphasis is laid on the painstaking apprenticeship necessary to acquire the technique of this admittedly difficult operation. Its advantages to the patient, however, are so outstanding that it has already become the operation of election for the large majority of patients suffering from an obstructing prostate. It is worthy of note, also, that Nesbit still advocates total perineal prostatectomy for the carcinomatous prostate, reserving the transurethral operation for those cases of cancer that are too extensive for such total removal.

It is both with entire satisfaction and pleasure that one recommends highly the careful study of this outstanding monograph by all surgeons and students who wish a clear exposition of the newest and most valuable method of relief of patients suffering from prostatism. The writer is deserving of many congratulations on having produced a work of such outstanding merit.

WILLIAM C. QUINBY

BOOKS RECEIVED

A MANUAL OF PULMONARY TUBERCULOSIS AND AN ATLAS OF THORACIC ROENTGENOLOGY. By David O. N. Lindberg, M.D., F.A.C.P., Lecturer on Tuberculosis, State University of Iowa, College of Medicine; Director of Roentgenology, State Sanatorium, Iowa. Cloth. Price, $6.50. Pp. 233, with 289 illustrations. Springfield, Illinois: Charles C Thomas, 1943.

THE ARTHROPATHIES: A HANDBOOK OF ROENTGEN DIAGNOSIS. By Alfred A. de Lorimier, A.B., M.A., M.D., Colonel, Medical Corps, United States Army; Commandant, The Army School of Roentgenology, Memphis, Tenn.; Formerly Director, Department of Roentgenology, Army Medical School, Washington, D. C. Cloth. Price, $5.50, Pp. 319, with 678 illustrations. Chicago: Year Book Publishers, Inc., 1943.

OUTLINE OF ROENTGEN DIAGNOSIS: AN ORIENTATION IN THE BASIC PRINCIPLES OF DIAGNOSIS BY THE ROENTGEN METHOD. By Leo G. Rigler, B.S., M.B., M.D., Professor of Radiology, University of Minnesota, Minneapolis, Minnesota. Fabricoid. Price, $6.50. Pp. 196, with 254 illustrations. Philadelphia: J. B. Lippincott Company, 1943.

ENCEPHALOGRAPHY. By E. Graeme Robertson, M.D. (Melb.), M.R.C.P. (Lond.), F.R.A.C.P., Physician to Out-Patients Royal Melbourne Hospital; Physician, Austin Hospital for Cancer and Chronic Diseases; Physician, Victorian Eye and Ear Hospital; Visiting Neurologist, 115 Australian General Hospital, Heidelberg; Neurologist to the Talbot Colony for Epileptics; Clinical Assistant, Children's Hospital, Melbourne. Monographs from the Walter and Elia Hall Institute of

Research in Pathology and Medicine, Melbourne, Number 2. Boards. Price, $5.00; 25s. Pp. 105, with 68 illustrations. New York: G. E. Stechert & Co.; Melbourne & London: Macmillan, 1941.

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DEPARTMENT OF TECHNIQUE

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AN ACCESSORY DEVICE FOR SPOT FILM ROENTGENOGRAPHY

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ESQUIMALT, B. C.

THE usefulness of securing multiple spot roentgenograms for permanent record and further study of a lesion detected during roentgenoscopic examination of the gastrointestinal tract, particularly the stomach and duodenum, has been well recognized. The procedure is of value in the study of the duodenal bulb or a suspected area of infiltration of the stomach wherein multiple spot roentgenograms, secured at regular intervals, serve as a record of motor function of the barium-filled viscus and also provide the observer with several projections at various angles.

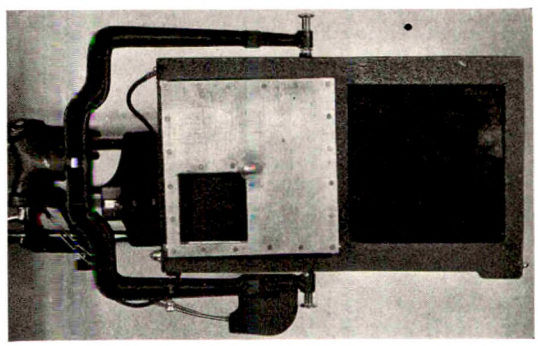


Fig. 2. The unit is shown assembled and ready for use in the spot film device.

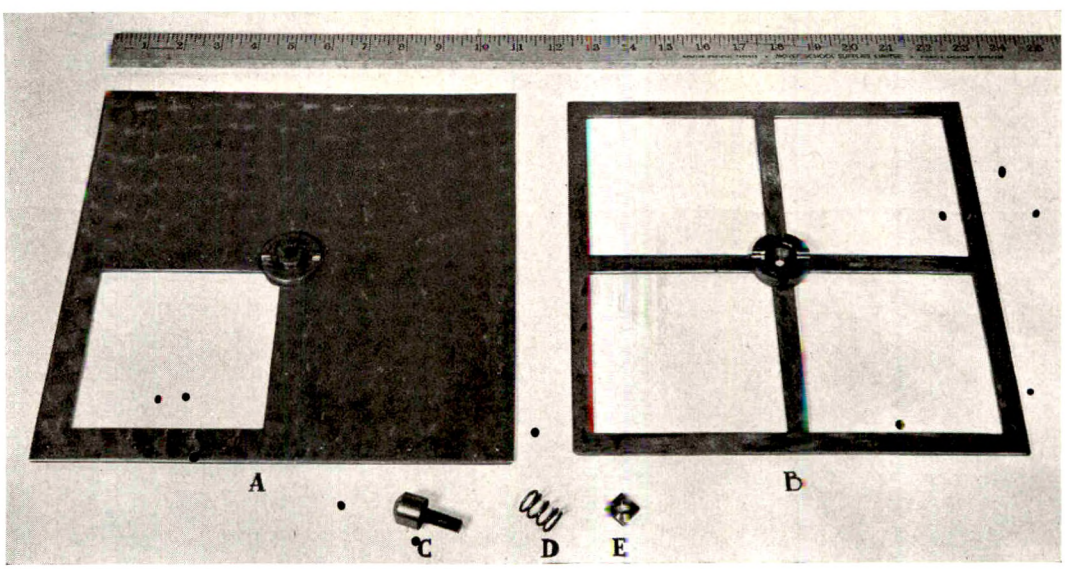


Fig. 1. This shows the components of the unit. *A*, rotating lead screen; *B*, frame; *C* central bolt; *D*, steel spring; *E*, nut.

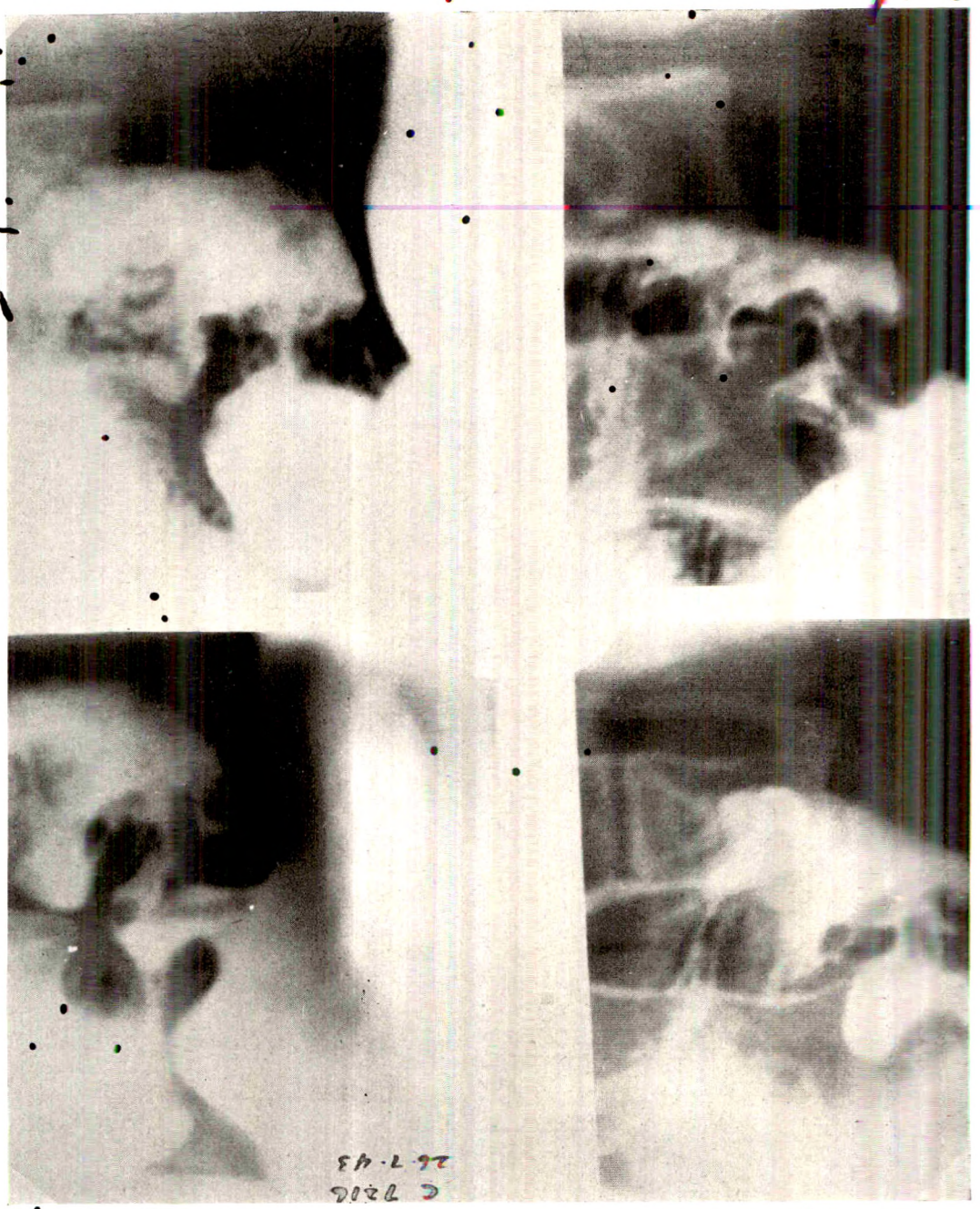


Fig. 3. Roentgenograms made with the technique described.

The time consumed in exposing several individual small films, together with the awkwardness of the procedure, have led to the construction of the device to be described, which allows four exposures on an 8×10 inch film. No originality is claimed, but a perusal of the literature has failed to discover the description of a similar device. It is known that certain manufacturers of roentgenologic equipment make

use of the principle by a different method; namely, rotation of the cassette within the spot film device after the introduction behind the fluoroscopic screen of a radiopaque metal sheet from which a corner, amounting to one-quarter of the area of an 8×10 inch film, is removed.

The device herewith described is adapted for the type of spot film device which does not utilize the cassette-rotation principle. It consists mainly of four items. The components of the apparatus are demonstrated in Figure 1, and the assembled device is demonstrated ready for use in Figure 2.

1. *The Frame:* (Fig. 1B). This portion of the unit is made of $\frac{1}{8}$ inch steel. It is composed of four equal squares at the center of which is a threaded hole. A ratchet is fitted around the central axis (see below). This frame is fitted to the slots on the back of the fluoroscopic screen where the compression device is ordinarily placed.

2. *The Rotating Lead Screen:* (Fig. 1A). This is constructed of $\frac{1}{16}$ inch sheet steel to which is riveted a similar sheet of $\frac{1}{16}$ inch lead. A corner, amounting to approximately one-quarter of the area of an 8×10 inch film and equalling the size of each of the squares of the frame, is removed. A *non-threaded* hole, slightly larger than the *threaded* hole of the frame is present at the center to permit free movement on the central bolt described below. A ratchet to fit its counterpart on the frame is built into the central portion around the axis.

3. *The Central Bolt:* (Fig. 1C). This is the axis on which the lead screen rotates; it is threaded into the hole in the center of the frame. This bolt is held in position by a nut (Fig. 1E) at the back of the frame and can be adjusted as required. The bolt has a hollow cap at the opposite end which accommodates:

4. *The Steel Spring:* (Fig. 1D). This serves to maintain sufficient tension between the frame and lead screen to allow free rotation of the latter, but enough to allow locking of the ratchet. The tension can be adjusted by the nut.

The two parts of the ratchet mechanism or interlocking device are each welded onto the adjacent surfaces of the frame and lead screen at their central axis. As shown in Figure 1, the ratchet component of the frame has four grooves placed at right angles to each other. Its adapting piece on the lead screen possesses two elevations which fit into the opposite two grooves on the frame. It follows that with the unit assembled, this arrangement permits rotation of the lead screen one-quarter turn for each slot in the frame.

OPERATION

When one has an area of interest for record, the unit is placed in the spot film device as shown in Figure 2 and the area to be recorded is centered to the square. An 8×10 inch cassette is then placed in the roentgenographic position in the spot film device and the exposure is made. On withdrawal of the cassette to its shielded position, the lead screen is rotated 90 degrees to the second position and the procedure repeated until four exposures have been made. A film exposed with the use of this device is shown in Figure 3.

Although the central beam is not centered to the square, this has led to no notable change in roentgenographic effect.

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ABSTRACTS OF ROENTGEN AND RADIUM LITERATURE

ROENTGEN DIAGNOSIS

SKELETAL SYSTEM

Batson, Oscar V. The rôle of the vertebral veins in metastatic processes. *Ann. Int. Med.*, Jan., 1942, *16*, 38–45.

Metastatic processes, either neoplastic or infectious, spread in ways that do not conform to the simple pattern of lymphatic or circulatory dissemination of such diseases as tuberculosis or erysipelas. For instance, the frequent brain abscesses secondary to lung abscesses do not follow such patterns. And the spread of prostatic carcinoma does not correspond to the pattern of the pelvic lymph circulation.

The author studied the question of the spread of prostatic carcinoma by making injections of roentgen opaque substance into the deep dorsal vein of the penis in animals. He found a network of valveless veins which he calls the vertebral vein system. It includes the veins of the wall of the trunk, which include the veins of the breast, the veins of the head and neck, the veins which supply the walls of the blood vessels of the extremities and the perivertebral veins. This system parallels the portal, caval and pulmonary venous systems and provides a by-pass around them. It also provides a pool of reserve blood when the body cavities are compressed, as these veins carry more blood than the regions supplied by them require. There are many connections between the vertebral and caval venous systems. This system therefore provides pathways for the spread of diseases between distant organs. For instance the ocular palsies which occur in incision abscesses of the abdominal wall may be caused by emboli reaching the skull through this system.

This system of veins should be examined at all autopsies in order to gain a more complete knowledge of it.—*Audrey G. Morgan.*

Smyth, C. J., and Freyberg, R. H. A study of the hereditary nature of gout; a report of two families. *Ann. Int. Med.*, 1942, *16*, 46–56.

It has long been known that gout is probably a hereditary disease but so far as the authors know no systematic study has ever before been made of the uric acid content of the serum in members of gouty families.

They have made such a study in the 29 individuals in the families of 2 patients with gout. The family trees are given and roentgenograms of the bones of the feet.

They found that 7 of the 8 adult males in these two families had an excess of uric acid in the blood and 5 of them had clinical gout. In one family the father and 3 sons had clinical evidence of gout. In the other a father with clinical gout had 2 sons with hyperuricacidemia but no clinical symptoms of the disease. None of the females in either of the families had any evidence of gout.

As gouty individuals so frequently show an excess of uric acid in the blood and urine the disease is thought to be due to a defect in purin metabolism. But it is not known just what the nature of the defect is nor is the real significance of the excess of uric acid in the blood understood.

A study of the blood uric acid in the families of patients with gout would help to clear up these questions. It would also help to detect gout earlier and save the patients much useless suffering in later life.—*Audrey G. Morgan.*

Reimann, Hobart A., Havens, W. Paul, and Herbut, Peter A. Hodgkin's disease with specific lesions appearing first in the skin. *Arch. Int. Med.*, Sept., 1942, *70*, 434–443.

A case is described in a man of forty-five in whom the first manifestation of disease was the appearance of bright red spots on the skin. At this time there were no demonstrable enlarged lymph nodes and the most probable diagnosis seemed to be chronic relapsing febrile, non-suppurative panniculitis. After some months a slight cough developed. There were no physical abnormalities in the chest. A roentgenogram showed round densities in the lower halves of both lung fields, suggesting new growths, and multiple clear areas which resembled areas of emphysema. Abnormal enlargement of the lymph nodes was never observed clinically and the diagnosis was not definitely established until two years later when the patient died and

necropsy showed the typical lesions of Hodgkin's disease in the abdominal lymph nodes.

This is one of the very few cases in which specific lesions of the skin appeared before other manifestations of Hodgkin's disease.—*Audrey G. Morgan.*

ROENTGEN AND RADIUM THERAPY

SCHMIDT, ERNST A. Incidence of multiple primary tumors and the problem of acquired cancer immunity. *Radiology*, August, 1942, *39*, 208–213.

Among 3,700 consecutive autopsies performed by the Department of Pathology of the University of Colorado Medical School from 1925 to 1940 there were 42 cases of multiple primary tumors. In 38 cases the patient had two tumors and in 4 three tumors. Tumors can be designated as multiple primary tumors only if it is reasonably certain that they have developed independently and have no direct or indirect connection with each other.

Among these cases a combination of benign tumors was found in only 2 cases; the most frequent combination was that of a benign and a malignant tumor which was seen in 27 cases.

The significance of these multiple tumors from the standpoint of immunity is discussed and the author concludes by saying that he believes that the rarity of multiple primary malignant tumors may be brought about by factors of acquired immunity and that in the future a knowledge of such processes may become important in the fight against cancer.—*Audrey G. Morgan.*

RITVO, MAX, HOUGHTON, JOHN D., and MCDONALD, EUGENE J. Cancer in childhood. *Radiology*, Sept., 1942, *39*, 278–282.

According to the census 153,846 persons died of cancer in the United States in 1939 and of these 1,103, or 0.7 per cent, were less than fifteen years of age. The vital statistics for Massachusetts for 1939 show that the death rate for cancer in childhood was greater than that for pertussis, pulmonary tuberculosis, measles, diabetes cerebrospinal meningitis, syphilis, scarlet fever or typhoid. This shows the seriousness of the problem of cancer in childhood.

The authors present a study of all the cases of cancer in patients less than fifteen years of age seen at the Boston City Hospital for the twenty-five year period from 1915 to 1939 inclusive. A table is given showing the nature of these tumors and a comparison with the reports of Kellert for a single county and Scotti for the New York Post Graduate Medical School. The total number of cases seen at the Boston City Hospital was 72. Of these, 23 were intracranial, 13 of the kidney and 11 of bone. During this same period 11 cases of Hodgkin's disease, other than sarcoma, were seen, and 27 of leukemia. The commonest tumors were glioma (medulloblastoma, astrocytoma and mixed gliomas), Wilms' tumor, osteogenic and Ewing's sarcoma and various types of soft tissue sarcomas. There were 3 cases of carcinoma of the acute type. There were only 2 cases of neuroblastoma of the adrenal, which is a very small number as compared with that in series reported previously. Among the earliest cases of cancer were one of Wilms' tumor in a patient one year of age, medulloblastoma in patients one and two years of age and a fibrosarcoma in a child two years of age. There was only one congenital tumor in the series—a malignant teratoma of the orbital region.—*Audrey G. Morgan.*

SHOULDERS, H. S., TURNER, E. L., SCOTT, L. D., and GRANT, W. H. Effect of combined fever and x-ray therapy on far-advanced malignant growths. *Radiology*, 1942, *39*, 182–193.

As early as 1935 Warren found that fever therapy increased the effectiveness of irradiation for malignant tumors. The work done on the subject since that time is reviewed and the authors report their combined fever and radiation therapy in 42 cases of far advanced inoperable malignant tumors of various types in the tumor clinic of George W. Hubbard Hospital of Meharry Medical College. They describe the technique in detail and give a table showing the types of cases treated and the results. They also describe in detail 2 cases of osteogenic sarcoma, 1 bronchogenic carcinoma, 1 ulcerating scirrhous carcinoma of the breast and an adenocarcinoma of the urinary bladder.

They have had no mortality attributable to the treatment. There was symptomatic improvement in 27 of the 42 cases (65 per cent). In some cases there was unquestionable evidence of regression of the primary growth and of metastases. They believe there are greater possibilities in the use of combined fever and radiation therapy than in irradiation alone. They believe that they have had the most satis-

factory results from this combined form of treatment in sarcomas.

Though it is impossible as yet to pronounce any judgment in regard to cure they believe that their results in far advanced inoperable cases justify a trial of this association of methods in cases that are still operable. It is possible that surgical results might be improved. They hope that other workers will test their results.—*Audrey G. Morgan.*

Schreiner, B. F., and Christy, C. J. Results of irradiation treatment of cancer of the lip; analysis of 636 cases from 1926–1936. *Radiology*, Sept., 1942, *39*, 293–297.

The records of the New York State Institute for the Study of Malignant Diseases from Jan. 1, 1926, to Dec. 31, 1936, showed 636 consecutive cases of cancer of the lip treated by irradiation. Cancer of the lip occurred about 38 times as frequently in men as in women and about 29 times as frequently on the lower as on the upper lip. The youngest patient was twenty-eight and the oldest ninety-two years of age. The use of tobacco was very common. There were only 15 cases of leukoplakia and only 3.6 per cent of the cases were positive for syphilis.

The authors found irradiation satisfactory in the treatment of the primary lesion but in the majority of cases with metastases the results are still unsatisfactory. They urge the use of both irradiation and surgery in an attempt to improve the results in cases with metastases.

For the whole group of 636 cases the absolute five year survival rate was 58.9 per cent. But if those cases are counted out in which the patient died of intercurrent disease and was free of recurrence at the time of death and those that were lost track of before the end of the five year period the cure rate is 74.4 per cent. Of the 27 patients in whom metastatic lesions were positively proved and who were treated with radon seed and radium needle implantation and with high voltage roentgen rays only 2, or 7.4 per cent, remained well five years or longer. None of the 11 patients with proved metastatic lesions who were treated with high voltage roentgen rays or radium pack alone survived for as long as five years. Ten patients had metastases diagnosed clinically but not proved histologically. They were treated with radon seed and radium needle implantation and external irradiation. Nine of these were in Grade 2; 5 died of the disease and 4 are living and well. The other patient, in Grade 3, is alive and well. Nine patients in Grade 3 with metastases diagnosed clinically but not proved histologically, treated with high voltage or roentgen radiation or radium pack alone, are alive and well.—*Audrey G. Morgan.*

Soley, Mayo H., and Stone, Robert S. Roentgen ray treatment of hyperthyroidism. *Arch. Int. Med.*, Dec., 1942, *70*, 1002–1016.

Roentgen treatment of hyperthyroidism has been somewhat neglected in recent years. Therefore the authors report a series of 43 cases treated in this way. Details of the technique are described and tables given showing the results. Twenty-two of the patients were given iodine in conjunction with the roentgen treatment while 21 were not.

The results show that roentgen irradiation reduces thyroid tissue sufficiently to produce the same results as surgical subtotal thyroidectomy. Patients with nodular goiter should have the nodules removed surgically. The best patients for roentgen treatment are those with marked degrees of exophthalmos.

Among the 43 patients 25, or 58.1 per cent, were freed of clinical symptoms in an average time of 8.7 months, while 8, or 18.6 per cent, were markedly improved. In both these groups the thyroid decreased to normal size and basal metabolism fell to normal. Three patients were not followed up. Two patients with congestive heart failure died before treatment could be completed. Four were operated on because of failure of roentgen treatment or for other reasons. One patient was given a single course of treatments as preparation for subtotal thyroidectomy.

The most serious complication was tracheitis and esophagitis, which occurred in 15 cases. This, however, was probably due to the fact that treatment was at first given from two anterior fields, resulting in cross fire on these organs. Since this method has been changed and irradiation is given from only one anterior field these complications have not occurred.—*Audrey G. Morgan.*

Hunter, A. F. Roentgen therapy of hypertrophic scars and keloids. *Radiology*, Oct., 1942, *39*, 400–409.

Keloids are benign overgrowths of connective tissue caused by traumatic, infectious, chemical or physical agents. It is not always easy to dif-

ferentiate between keloid and hypertrophic scar but keloids progress with time while hypertrophic scars tend to regress.

The author discusses 931 patients with keloids or hypertrophic scars treated in the Department of Radiology of the Presbyterian Hospital, New York, from 1922 through 1941. Follow-up data were lacking in 258 of these, leaving 673 to be considered. Of these 588 were treated by roentgen therapy alone and 85 by roentgen therapy and surgery. Tables are given showing the details of the results and photographs of a number of cases given before and after treatment. In the great majority of cases given roentgen treatment, 491 out of 588, the scars became softer, flatter and less conspicuous without other treatment. As a rule, fresh scars respond to irradiation better than old ones. Preoperative irradiation may enable the surgeon to operate in otherwise inoperable cases. It is still an open question as to whether preoperative and postoperative treatment is better than postoperative treatment alone.

It is hoped that further study will show a basic biologic difference in the radiosensitivity of keloid and hypertrophic scar tissue. The irradiation of cultures of the two types of tissue may prove helpful in this study.—*Audrey G. Morgan.*

Sims, J. L., and Carns, M. L. Post-radiation panmyelophthisis clinically simulating agranulocytosis. *Ann. Int. Med.*, Dec., 1942, *17*, 1021–1028.

A case is described in a woman of forty-three admitted to the Wisconsin General Hospital September 7, 1939, complaining of a mass in the abdomen. Blood count was 4,740,000 red cells and 9,400 white cells with 91 per cent neutrophils, 7 per cent lymphocytes and 2 per cent monocytes. Roentgen examination showed pleural effusion and enlargement of the mediastinal lymph nodes and partial atelectasis of the left lower lobe. A biopsied lymph node showed lymphosarcoma. Photomicrographs of lymph nodes and vertebral marrow are given.

From September 15 to 29 irradiation was given, not more than one area being treated each day. One-fourth of the body surface was exposed at a time, a dose of 50 r being given twice to each area in turn and then 200 r given to each of three abdominal preaortic areas and to the splenic region. On September 27 the white blood cell count was still 6,900 but on September 29 it fell suddenly and continued to fall. The patient died October 8, twenty-three days after the beginning of radiation therapy. The leukocyte count October 7 was 75. The last red cell count October 4 showed 4,735,000 red cells and on October 7 the red cells still appeared normal in smears, but there was some decrease in the number of platelets.

Autopsy showed such extensive and severe damage of bone marrow as to amount to almost complete aplasia of all the hematopoietic tissue.

It is probable that in this case the relatively longer life span of the red cells and their greater resistance masked the damage to the tissue that produced them and that this was really a case of panmyelophthisis rather than one of agranulocytosis.

A study of the literature does not show any cases of true agranulocytosis caused by irradiation. The cases of apparent severe damage to white cells alone seem to be manifestations of generalized damage to the marrow. A given amount of radiation apparently affects all cell types and the peripheral picture depends on the longer life span of the circulating red cells and the lesser degree of radiosensitiveness of the erythropoietic elements.—*Audrey G. Morgan.*

Newell, R. R., Falconer, Ernest H., Hill, H. P., Lawrence, John H., Wood, David A. and Wyckoff, Harry. Panel discussion on the leukemias and lymphoblastomas. *Radiology*, Sept., 1942, *39*, 298–305.

This article reports a panel discussion of the leukemias and lymphoblastomas, diseases affecting the blood-forming organs and the lymphatic system. It was generally agreed that the etiology of these diseases is unknown and classification is therefore difficult. A considerable part of the discussion was devoted to treatment and here, too, there was agreement that it is hopeless, so far as real cure is concerned. It is symptomatic and judgment as to the effects must be based on the patient's condition rather than on the decline in the white cell count. Spontaneous remissions occur in a certain percentage of cases so it is difficult to judge the effects. The possible treatments are medical, with arsenic, and irradiation. Dr. Falconer is an ardent advocate of arsenic treatment with large doses and the details of his technique are described. There was a discussion of the real nature of the action of arsenic and irradiation and whether the leukemias are really neoplastic diseases.

Dr. Lawrence advocated the use of small doses of radiophosphorus in leukemia, a dose equivalent to 4 to 8 roentgens whole body irradiation per day for four to eight weeks. He does not use local irradiation at all. Radiophosphorus may be used even if there is anemia, but the dose must be smaller. Radiophosphorus is dangerous unless carefully controlled.

It was agreed that Hodgkin's disease is probably a generalized disease even from the first. Dr. Newell advocated the use of small doses of irradiation, beginning with doses of 50 roentgens rather than 250. Dr. Falconer believed that the smaller the dose that could be used to relieve the patient's symptoms, the better. The severity of the disease does not run parallel with the size of the tumors. A combination of arsenic and roentgen treatment in the leukemias seems to give better results than either alone.—*Audrey G. Morgan.*

Kerr, H. Dabney. Irradiation treatment of cavernous hemangioma with special reference to so-called contact roentgen irradiation. *Radiology*, Oct., 1942, *39*, 383–388.

Some authors hold that cavernous hemangiomas are self-limited and may even regress spontaneously, but the author has seen enough rapidly spreading lesions in infants to make him believe that they should be treated as soon as detected. Small superficial lesions up to 7 to 8 mm. in diameter may be treated with carbon dioxide snow but larger ones should be treated by irradiation.

He gives tables showing the results in two series of cases. The first series consisted of 96 cases treated with radium plaques, 5 to 7 mg-hr. per sq. cm. being given the lesion at a distance of 2 mm. The results were good in 63.5 per cent of the cases, fair in 15.6 per cent, poor in 11.6 per cent, and 10.3 per cent were inadequately treated or followed up. The other series consisted of 49 cases treated with roentgen rays at a short distance by means of the Chaoul contact unit. With an intensity of about 120 roentgens per minute at 5 cm. distance or 340 r per minute at 3 cm. distance, 300 to 500 r was given to the lesion. In this series the results were good in 69.3 per cent, fair in 14.2 per cent, poor in 6.1 per cent, and 10.2 per cent were inadequately treated or followed up.

The slightly better results obtained with roentgen irradiation are probably not of great significance but the author emphasizes the fact that in his hands short distance roentgen irradiation has given at least as good results as treatment with radium plaques.—*Audrey G. Morgan.*

Orndoff, Benjamin H. Roentgen therapy in the management of some non-malignant diseases affecting the organs of the female pelvis. *Radiology*, August, 1942, *39*, 127–134.

Hyperplasia and tumors of the endometrium and myofibromata seem to be a rather closely related group of diseases of the tissues of the female pelvis apparently brought about by a common factor, estrin. The author's theory is that a predominance of the action of estrin and a lack of that or progesterone is brought about by the long intervals between pregnancy in modern life.

Roentgen examination is of the greatest value in both the diagnosis and treatment of these conditions. It is believed that an irradiation menopause interferes less with later sexual life than a surgical menopause. If a temporary interruption of menstruation is desired, radium should be used because of its more intense action on the uterine mucosa and its lesser action on the ovary. If permanent castration is necessary roentgen rays are to be preferred because of their greater action on the ovary. It is generally agreed that about 300 r delivered into the pelvis is required to bring about cessation of menstruation at the beginning of the fourth decade of life. If radium is used about 500 mg-hr. should be applied to the endometrium with the usual filter and plain applicator. Of course the details of technique must be considered in each individual case. There has been a gradual change from surgery to irradiation in the treatment of these non-malignant conditions.

In the discussion several physicians disagreed with Dr. Orndoff's theory that a practically continuous state of pregnancy from maturity to the menopause would benefit the health of women.—*Audrey G. Morgan.*

Kaplan, Ira I. Radiation in cancer of the corpus uteri. *Radiology*, 1942, *39*, 135–143.

Wertheim's operation has practically been given up in favor of irradiation in the treatment of cancer of the cervix but until recently surgery has been thought to be indicated in cancer of the body of the uterus. The author reports a series of 101 cases treated at Bellevue Hospital, New York, which seem to indicate that this is

no longer the case. The policy at Bellevue Hospital has been to irradiate operable carcinomas of the body of the uterus followed by hysterectomy six to eight weeks later. In inoperable cases irradiation alone is used. The best method of irradiation is by the intrauterine application of radium, and part of the success in recent cases has been due to the use of better applicators. He describes and illustrates a ring applicator by means of which the radium can be applied at the desired points as shown on a roentgenogram.

Of the 101 cases 5 proved to be non-malignant and 1 was not definitely proved to be malignant. Among the 95 patients with definitely malignant lesions 34 are known to be dead. Among the 35 patients known to be living 7 have lived one year, 4 two years, 7 three years, 5 four years, 1 six years, 2 seven years and 1 each eight, nine, ten, eleven, thirteen and fourteen years. The 3 cases treated with the radium ring are too recent for a report of results. One was treated by roentgen irradiation in 1923 for benign bleeding confirmed by biopsy and later returned with recurrent bleeding from a carcinoma also confirmed by biopsy.—*Audrey G. Morgan.*

REINHARD, M. C., GOLTZ, H. L., and SCHREINER, B. F. A study of radiological treatment of cancer of the cervix. *Radiology*, August, 1942, *39*, 144–150.

This article presents a study of 557 cases of cancer of the cervix treated in the New York State Institute for the Study of Malignant Diseases from 1931 to 1935 inclusive. It represents all the cases of Stages I, II and III according to the League of Nations Classification. The Stage IV cases were not included. Graphs are given showing the details of the results. The age graph shows a definite peak at 47.5 years.

Among the 557 cases, 457 were classified into two groups depending on whether they were given only primary irradiation or whether in addition to this they were given further irradiation. About half required additional treatment. The survival rate in this group is less than in the group that required only primary irradiation. This might be due to the nature of the disease itself, extension of the disease to regions where adequate dosage was not given, normal differences in individuals or the age of the patient. Survival was better in the older age groups. Isodose and dosage distribution curves are given. Survival is progressively poorer with increased extent of the disease. Cases with only primary irradiation varying from 4,000 to 8,000 r showed 75 per cent survival free of disease in Stages I and II and 55 per cent in the Stage III cases. It is probable that the poorer survival rate in Stage III cases is due to the fact that the disease has spread to regions that are not given adequate dosage and it therefore seems probable that dosage to the lateral parts of the pelvis should be increased. Among the patients in all three stages who were given additional irradiation the highest survival was in patients who were given less than 2,000 r in addition to the primary irradiation.—*Audrey G. Morgan.*

WALLACE, WILLIAM S. The intestine in radiation sickness. *J. Am. M. Ass.*, Feb. 15, 1941, *116*, 583–586.

In view of the fact that the colon has frequently been studied with the intention of finding the cause of the diarrhea seen in radiation sickness and the findings have been inconclusive, and as diarrhea has been shown to be frequently due to disease of the small intestine, the author studied 10 patients with carcinoma of the cervix. The small intestine was examined before and after a protracted series of high voltage roentgen treatments. In most cases the rays were generated at 400 kv., using 4 mm. copper filter with a half-value layer of 5 mm. of copper. Three of the patients were each given about 5,000 roentgens intravaginally, using 200 kv. and filter of 1 mm. of copper, while all received between 1,700 and 2,200 r from each of six 10 by 15 cm. fields, around the pelvis. Before treatment, the small intestine in each case presented essentially the normal appearance previously described by various workers.

At the end of the protracted series of therapy in no case was there any demonstrable change in the duodenum or jejunum, both of which lay almost entirely outside the irradiated areas. The two most common changes were segmentation of the barium stream and flattening of the pattern of the ileal mucosa. Segmentation was observed in all but 1 case and changes in pattern, varying from a slight to a marked degree, were found in 8 of the 10 cases. A decrease in motility was shown in 7 instances, and an absolute diminution in the diameter of the ileal lumen due to spastic contraction was shown in an equal number. There was no apparent quantitative variation in effect obtained by slightly more than 20 per cent variation in dosage, by

the use of 200 kv. and 1 mm. of copper as compared to 400 kv. and 4 mm. of copper, or by the supplementary use of a heavy dose of intravaginal irradiation at 200 kv. and 1 mm. of copper. Early in his investigation the author conceived the opinion that the syndrome of radiation sickness might be due to a failure of the impaired mucosa to absorb food materials, with the production of an actual deficiency state, but this does not seem to be true when therapeutic doses of radiation are used. It appears likely that the diarrhea from deep pelvic irradiation is due to changes in the large rather than in the small intestine, as all demonstrable changes in the latter would tend to produce an opposite effect.—*S. G. Henderson.*

Alyea, Edwin P., and Henderson, A. F. Carcinoma of the prostate. *J. Am. M. Ass.*, Dec. 5, 1942, *120*, 1099–1102.

Within the last two years a new approach to therapy for carcinoma of the prostate has been offered. The authors present a brief report of the immediate results of castration in 40 cases of carcinoma of the prostate. What the later results will be they do not attempt to prophesy. The immediate result with all these patients was almost miraculous. Their general well being and energy at once improved, their appetites picked up and they gained weight. One of the striking features of their improvement was the rapid relief of metastatic pain, usually within twenty-four to forty-eight hours after castration.

Castration was performed on 14 patients who showed no evidence of metastasis. It was hoped by this, in addition to causing regression of the primary growth, to retard or prevent the spread of the tumor. Seven of these have had roentgen examination recently and as yet none show metastases.

The increase of serum acid phosphtase in carcinoma of the prostate is most interesting. Acid phosphatase is found in largest amounts in the adult prostatic epithelium and carcinoma of the prostate, which is a tumor composed of this cell. When metastasis takes place, the tumor cells are expressed into the blood stream and lymphatics and cause a rise in the serum acid phosphatase. Therefore this is an indication of tumor or adult prostatic epithelium activity. This series shows that of 17 patients with metastasis 10 had normal serum acid phosphatase and 7, elevated.

Clinical evidence of regression of the tumor is indicated in the patients' relief of obstruction. Rectal examination in every 1 of the 30 patients who returned for examination showed considerable reduction in size and softening of the gland. Further direct evidence of improvement is shown in the clearing of the urinary infection which most of these patients have before and after transurethral resection.

The usual evidence of metastasis of carcinoma of the prostate is seen in the bones of the lumbar spine and pelvis. Following castration there is a striking change in the picture. There is a distinct smoothing and sclerosing of bone detail approaching more normal type bone. The reaction is similar to that seen in post-irradiation healing. The authors also found regression of pulmonary metastatic lesions in 4 patients following orchiectomy. In 2 patients the metastatic nodules disappeared completely.—*S. G. Henderson.*

Creevy, C. D. The diagnosis and treatment of early carcinoma of the prostate. *J. Am. M. Ass.*, Dec. 5, 1942, *120*, 1102–1105.

The incidence of carcinoma of the prostate is increasing and its prognosis is bad. This rise seems to be due, not to a heightened individual susceptibility to cancer, but to a striking increase in longevity. Despite this increase, early prostatic carcinoma continues to be a clinical rarity.

Prostatic carcinoma causes no symptoms until it interferes with urination or produces pain by local extension or metastasis. Three-fourths of the cases arise in the posterior lamella at a relatively great distance from the urethra; hence obstruction develops late. The average prostatic carcinoma grows slowly. About 8 per cent of prostatic cancers develop within spheroids of benign hypertrophy which hide them from the examining finger. Most elderly men regard a certain amount of urinary difficulty as an inevitable accompaniment of advancing years. Many physicians are not thoroughly aware of the serious significance of isolated hard nodules in the prostate. The earliest carcinoma of the prostate which is recognizable clinically appears as a small stony hard, irregular, poorly circumscribed nodule. Not all hard nodules in the prostate are cancerous, and careful study may be required to determine their nature. If the nodule persists, biopsy is indicated, and it is here that aspiration biopsy should be of great

value. In the author's experience, biopsy can be done satisfactorily in early cases only through a perineal exposure under direct vision.

Reactions to radiation therapy are often severe and authenticated cures few and far between. Castration as developed by Huggins is, and will continue to be, very difficult to justify to the patient without symptoms, and it seems to have no place at present in the treatment of early cases without pain although often brilliantly successful in late cases as a palliative measure. The use of estrogens in the early cases appears to hold great promise, and it demands long and painstaking study. Like castration, it has produced striking results in late cases. Transurethral resection, despite its immense value as a palliative measure, cannot be mentioned as offering any hope of cure. The only method which can be said at present to offer a definite prospect of cure is radical perineal prostatectomy, so long advocated by Young. The mortality is as high as 10 per cent. There is a very definite risk of incontinence, permanent incontinence having resulted in 2 of 10 cases at the University Hospital. The chief obstacle to the satisfactory use of radical perineal prostatectomy is its low rate of applicability, which can be overcome only by a campaign of education designed to lead to early detection of prostatic carcinoma. The writer believes this operation is worthy of a widespread trial, especially since it carries a better chance of cure than any other method.—*S. G. Henderson.*

THOMPSON, GERSHOM J. Transurethral resection of malignant lesions of the prostate gland. *J. Am. M. Ass.*, Dec. 5, 1942, *120*, 1105–1109.

Because of the nature of malignant disease of the prostate gland, the patient generally is free of symptoms until the lesion is rather well advanced. Most surgeons who have advocated radical perineal prostatectomy have freely admitted that the procedure can be utilized in only 5 per cent of cases or less. Baron and Angrist studied the incidence of occult adenocarcinoma of the prostate gland among men beyond the age of fifty. They found that carcinoma of the prostate gland cannot be detected early enough by any clinical method of examination now known to make it possible to effect cure by radical surgical extirpation.

During the past ten years at the Mayo Clinic the author has not employed radical perineal prostatectomy. The evidence at hand clearly indicates that when carcinoma of the prostate gland has developed sufficiently to cause urinary obstruction the disease has spread through the perineural lymphatic vessels and cannot be completely eradicated by any surgical procedure. It is Thompson's present attitude that transurethral resection which adequately removes all the obstructing tissue, combined with bilateral orchiectomy (or, if the patient refuses this, irradiation or the administration of diethylstilbestrol), offers the patient a far better prognosis than does any radical procedure. This scheme of treatment is accompanied by much less risk to life than is the radical operation and offers almost certain assurance that control of micturition will be perfect.

In defense of this attitude the author presents a statistical study of a group of 887 cases of malignant neoplasms of the prostate gland in which transurethral resection was performed during the interval 1924 to 1941 inclusive. This group of 887 is composed only of patients who had definite symptoms or urinary obstruction. Transurethral resection was performed with either the Braasch-Bumpus, Stern-McCarthy or Thompson resectoscope. The removal of all obstructing tissue was the aim of the surgeon in each case. Ease and comfort in voiding, with excellent immediate control, is the rule. Only exceptionally is there any urgency, and very rarely does permanent incontinence result.

In practically all cases (858 of the 887, or 97 per cent) the neoplasm was adenocarcinoma. In 22 cases, or 2.5 per cent, the lesion was squamous cell epithelioma and in 7 it was mixed and squamous cell carcinoma. In approximately 56 per cent of the cases the lesions were of Grades 3 and 4.

Of the 877 patients who survived operation, 206, or 23.5 per cent, were irradiated. With the exception of a few instances in which radon seeds were implanted into the prostatic capsule through the perineum, treatment was by roentgen rays. Of 69 patients who received treatment prior to 1937, 10, or 14.5 per cent, have lived five or more years. Thompson analyzed a group of 337 patients who were operated on prior to 1937. Three of these patients died in the hospital. Of the 334 patients, 320 were traced and a total of 46 patients, or 14.1 per cent, were found to have lived five years or more after leaving the hospital. No patient who had a tumor of Grade 4 survived operation for as long as five years. No claim to cure is made in any case, and all the operations were done purely as palliative procedures.—*S. G. Henderson.*

Nesbit, Reed M., and Cummings, Robert H. Prostatic carcinoma treated by orchiectomy. *J. Am. M. Ass.*, Dec. 5, 1942, *120*, 1109–1111.

The authors present data on 75 cases of prostatic carcinoma treated by orchiectomy at the University Hospital and observed for periods of at least six months following that procedure. Eleven cases of 28 so examined (39 per cent) showed elevated serum acid phosphatase levels of 6 King-Armstrong units or higher. In 42 of the cases pain of metastatic origin was complained of on admission. Five obtained no relief whatever following operation, while 27 obtained complete relief of pain and 10 obtained partial relief. Eighteen patients complained of significant weight loss on admission to the hospital. Thirteen of these regained their weight losses. Thirty-eight additional patients have had appreciable gain in weight since orchiectomy. Nine patients at the time of orchiectomy complained of significant difficulty with urination. All these patients have experienced more normal urination since orchiectomy. Forty-one of the 75 cases have been examined rectally at least six months after operation. Twenty-seven cases showed definite regression in the size of the prostate gland although still recognizable as carcinoma to palpation. Eight cases have shown a regression in size so that the prostate is now atrophic, firm and smooth with no obvious infiltration. The prostates in 6 cases have changed from a condition which was obviously malignant before operation to a consistency now unrecognizable as neoplastic by rectal examination. Positive roentgen evidence of skeletal metastases was found in 31 cases. In 12 of these a roentgen examination was made at least six months following orchiectomy. Four show definite regression of metastases, while 8 show an increase in the osseous changes.

In summarizing, the authors state that in 48 cases pronounced clinical improvement has occurred. These patients have all shown regression of the primary neoplasm, have all gained weight and all who had pain prior to orchiectomy are now free from it and all are voiding satisfactorily. Seven additional patients have had complete relief of symptoms but there are no objective evidences of improvement either in the primary neoplasms or in the roentgenograms. There have been 20 failures. Four patients have died of carcinoma and 1 from congestive failure and infection. Fifteen living patients are considered to be failures, 5 have shown no favorable response at any time following operation, while 10 are considered delayed failures, having enjoyed freedom from all subjective evidences of their disease for periods of three to twenty-two months before symptoms recurred after gonadectomy. Regardless of the possibility of failure in some cases it is evident that gonadectomy is a worthwhile procedure in advanced prostatic malignancy and should be recommended when the diagnosis is established. —*S. G. Henderson.*

Gutman, Alexander B. Serum "acid" phosphatase in patients with carcinoma of the prostate gland. *J. Am. M. Ass.*, Dec. 5, 1942, *120*, 1112–1116.

While investigating the source of "acid" phosphatase in urine, Kutscher and Wolbergs in 1935 discovered that normal human prostate tissue is extremely rich in an "acid" phosphatase. The enzyme does not appear in appreciable amount in human or monkey prostate tissue until puberty. Prostatic "acid" phosphatase is believed to have some distinct physiologic function, but the precise nature of this function is not known.

Apart from occasional highly undifferentiated prostatic tumors which do not elaborate the enzyme, carcinomatous prostate tissue contains large amounts of "acid" phosphatase. This holds not only for tumor tissue at the primary site but also for distant metastases. When carcinomatous prostate tissue metastasizes, invasion of lymph or blood channels is accompanied by escape of the prostatic secretion into the circulation. Because of its high "acid" phosphatase content, the prostatic secretion present in blood can readily be detected by means of appropriate chemical methods for estimating the "acid" phosphatase activity of blood serum, which is increased by influx of the prostatic enzyme. The high "acid" phosphatase content of most prostatic carcinomas indicates that these tumors are not composed of functionally embryonal cells (since immature prostatic tissue is virtually devoid of the enzyme) but represent a type of malignancy made up of physiologically mature epithelial tissue.

Normal human serum contains very small amounts of "acid" phosphatase, less than 2.5 units activity per 100 cc. of serum as determined by an adaptation of the King and Armstrong method for "alkaline" phosphatase. This normally occurring enzyme is not of pro-

static origin. Elevated values, for the most part higher than encountered in any other disease, were obtained in about 85 per cent of a total of 177 cases of proved or suspected metastatic prostate carcinoma. About 90 per cent of patients with prostate carcinoma but without roentgenographic evidence of bone involvement gave values consistently less than 3 units, as did all normal subjects, all patients with prostatic disease other than carcinoma and more than 90 per cent of a total of 853 cases of nonprostatic disease. There is no general agreement at present as to the critical "acid" phosphatase level of the serum which may be accepted as diagnostic of prostate carcinoma with metastases. However, follow-up studies of any one patient with prostate carcinoma indicate that the development of metastases is accompanied by a rise in serum "acid" phosphatase above normal levels and that subsequent spread of secondary involvement is accompanied by further increases in serum "acid" phosphatase activity.

The increase in serum "alkaline" phosphatase is of osseous origin and reflects the extent and vigor of the osteoplastic reaction of bone at the site of skeletal metastases; but this rise is unspecific, being common to conditions characterized by widespread, accelerated osteoblastic activity or by obstruction of the biliary tract. The increase in serum "acid" phosphatase, on the other hand, is a more specific and constant manifestation of metastasizing prostate carcinoma.

Clinical Applications of the Serum "Acid" Phosphatase Determination. The method is incapable of detecting prostate carcinoma which has not yet metastasized. The determination is often particularly helpful when interpretation of roentgenograms as regards the presence of metastases is inconclusive. Differentiation of Paget's disease from osteoplastic metastases secondary to prostate carcinoma is facilitated by the test. A definitely and consistently increased serum "acid" phosphatase level has been found to be a generally reliable contraindication to radical prostatectomy. With the introduction of treatment by androgen control, the determination of serum "acid" phosphatase has become an almost indispensable guide to the selection of patients for castration, the regulation of estrogen therapy and the critical evaluation of results with these several forms of treatment. Various observers have noted an early precipitous fall in serum "acid" phosphatase, often seen after the first twenty-four hours following treatment by castration. As regards the effect of estrogens on the serum phosphatases in patients with metastatic prostate carcinoma, only meager data are available. These make it clear, however, that estrogens in appropriate dosage generally cause a fall in serum "acid" phosphatase and a delayed rise in serum "alkaline" phosphatase.—*S. G. Henderson.*

MISCELLANEOUS

MILLER, EARL R. A simple foreign body localization device applicable to standard fluoroscopes. *Radiology*, Oct., 1942, *39*, 464–465.

When the author tried to use the methods of foreign body localization, previously described, on his G. E. fluoroscopic table he found he could not use them without changing the design of the fluoroscope, changing the amount of shutter opening or getting special apparatus. Therefore he worked out a simple parallelogram system of foreign body localization which is described in detail and illustrated. It is a single tube shift method which can be used with any ordinary fluoroscope. The depth of the foreign body is given directly on a 4 to 1 scale. In using it with various test objects he has found that its accuracy is well within 5 per cent.—*Audrey G. Morgan.*

WARREN, SHIELDS. Blood findings in cyclotron workers. *Radiology*, 1942, *39*, 194–199.

Monthly blood counts were made on 34 workers of the cyclotron group of Harvard University and 51 of the Massachusetts Institute of Technology group. Forty-two were followed for over a year with the object of finding whether they were injured by the radiation. The results were for the most part negative. As with roentgen or radium radiation there is at first a transitory decrease in the white cell count followed by a rise. The fall is more marked in the lymphocytes than in the polymorphonuclear leukocytes. There were 4 individuals in whom there were quite marked variations in the white cell count. Graphs showing the findings in these cases are given. When slight irradiation causes marked variations in the white cell count it indicates that the subject has an unstable bone marrow and such persons should not be allowed to work with the cyclotron.

Red cell counts above five million and hemoglobin levels above 100 per cent were found in several cases. None of the group showed a persistent anemia and nucleated red cells were not found in any of them. Apparently adequate precautions are taken for the safety of these cyclotron workers.—*Audrey G. Morgan.*

Hodges, Fred J. The cyclotron as a medical instrument. *Radiology*, 1942, *39*, 440–453.

This was a lecture delivered before the Kalamazoo Academy of Medicine in honor of Augustus Warren Crane. It reviews the work done by physicists in developing the cyclotron for "atom-smashing" or the breaking down of atomic nuclei. The largest of these cyclotrons is located at the Crocker Radiation Laboratory at the University of California, Berkeley. By means of this instrument intense beams of high energy particles can be produced and manipulated easily and accurately. Natural radioactivity exists only in actinium, thorium and uranium. But by means of this method radioactive isotopes of all known elements can be produced.

These discoveries may be utilized in three ways in medicine: (1) experimentation with radioactive tracer substances in biology and medicine; (2) experimental use of artificially produced radioactive isotopes given internally for therapeutic purposes, and (3) the substitution of fast neutrons for roentgen rays and radium rays in external radiation treatment. The accomplishments in these fields so far are discussed. Intracellular radiation with radioactive isotopes seems to promise the production of selective ionization in the very tissues where ionization effects are desired. This has been tested in the treatment of leukemia with radioactive phosphorus with good results. The use of neutrons as a substitute for roentgen or radium therapy is still in the experimental stage but the results of the experiments are of intense interest to physicians and radiologists.—*Audrey G. Morgan.*

Aebersold, Paul C. The cyclotron; a nuclear transformer. *Radiology*, 1942, *39*, 513–540.

The cyclotron is generally called an atom smasher, but it is really a nuclear transformer, as its work is to transform the nuclei of the atoms, which are ten thousand times smaller than the atoms themselves, changing them into new forms. A technical review of the work of nuclear transformation is given, beginning with the work of Rutherford in 1919 who bombarded nitrogen with natural alpha particles and transformed the nitrogen nuclei into oxygen nuclei and continuing up to the work of E. O. Lawrence who conceived the idea of speeding particles up to very high energies for the bombardment of the nucleus by giving them successive accelerations, so that much lower voltages could be used instead of the much higher ones required to give the total acceleration at one push.

The different types of cyclotron from the first to the present one are illustrated and a photograph given of the giant cyclotron laboratory on a hilltop above the campus of the University of California. The building resembles an observatory which is used to look out at objects millions of times larger than man, while the cyclotron is used to investigate and work with infinitesimally small particles, millions of times smaller than he.—*Audrey G. Morgan.*

Hamilton, Joseph G. The use of radioactive tracers in biology and medicine. *Radiology*, Nov., 1942, *39*, 541–572.

Until the discovery of artificial radioactivity, the only way of studying metabolism was by chemical methods which have serious limitations as large amounts of the substance to be studied must be administered which frequently disturbs normal metabolic processes so the picture obtained may not be a true one. But now that radioactive isotopes of all the elements have been discovered, it is possible to "tag" the elements in the body with artificial radio-elements and their course through the body can be studied accurately and in detail.

There are three techniques for using radioactive isotopes of stable elements as tracers in the biological sciences: (1) the assimilation and elimination of the compound may be determined quantitatively by direct measurement of the radioactivity of samples of tissue after their removal from the body; (2) the selective accumulation of radioactive isotopes of several elements may be observed in the living animal by measurement of the radioactivity of the structures in situ, and (3) the distribution of the radioactive element may be studied by means of photographic films. The first is the commonest of these methods. The elements most intensively studied are phosphorus and radio-iodine. The former has proved valuable not only in biologic research but in the treatment of tumors:

and the leukemias while the latter has proved valuable in the treatment of hyperthyroidism.

The radioactive isotopes of only 21 elements have been studied and as each of the 92 elements has one or more isotopes the biological and therapeutic possibilities of this method of research are practically unlimited. The biological tracer research in metabolism must precede the therapeutic application of the element. —*Audrey G. Morgan*

LOW-BEER, B. V. A., LAWRENCE, JOHN H., and STONE, ROBERT S. The therapeutic use of artificially produced radioactive substances; radiophosphorus, radiostrontium, radioiodine, with special reference to leukemia and allied diseases. *Radiology*, Nov., 1942, *39*, 573–597.

Artificially radioactivated elements may not only be used as tags to follow the course of their inactivated isotopes through the body but as they emit rays similar to those given off by radium they may be used in treatment as substitutes for radium. The method of preparing and assaying radiophosphorus is described in detail. Naturally a millicurie of radiophosphorus is not comparable to a millicurie of radon in therapeutic effect. No means is known of applying radiation selectively to certain tissues, or of delivering a uniform amount of radiation of each cubic centimeter of tissue in the body.

Radioactive phosphorus has proved particularly effective in the treatment of chronic lymphatic and myelogenous leukemia, polycythemia vera and lymphosarcoma. Its value in the treatment of Hodgkin's disease has not yet been demonstrated. Nor has its value in the treatment of multiple myeloma, metastatic carcinoma and some other conditions been definitely established but as these diseases are inevitably fatal otherwise it is justifiable to experiment with its use. Illustrative cases of the diseases treated are described.

Chromium phosphate may prove of value in the treatment of primary and metastatic tumors of the liver and spleen and of neoplastic diseases of the reticulo-endothelial system as it concentrates selectively in this system. Radioactive strontium has a half-life of fifty-five days and emits rays of an energy similar to those of radiophosphorus. It concentrates selectively in the bones like calcium and may prove valuable in the treatment of primary and metastatic bone tumors. Radioactive iodine has proved valuable in the treatment of hyperthyroidism. —*Audrey G. Morgan.*

KENNY, JOHN M., and CRAVER, LLOYD F. Further experiences in the treatment of lymphosarcoma with radioactive phosphorus. *Radiology*, Nov., 1942, *39*, 598–607.

The authors have used radiophosphorus in the treatment of 22 patients with a proved diagnosis of lymphosarcoma. The patients were not selected as it was thought best to treat all stages of the disease in order to determine the value of the treatment. The greater danger in the use of radiophosphorus is damage to the bone marrow and therefore frequent blood counts were made during the treatment. The blood count was frequently decreased but the decrease was seldom serious.

Details of the results are given. Ten of the patients, or 46 per cent, are living; 12, or 54 per cent, are dead. Four of the patients had complete remission and have been free of disease for from three to twelve months. Two had recurrences which were controlled by further treatment; 3 had 75 per cent regression and 1 had about 50 per cent regression. Six of the living patients had had roentgen treatment before being given phosphorus; this had controlled the disease in 3 cases and partially controlled it in the other 3. Ten of the 12 patients who are dead were not helped at all by the phosphorus treatment. Eleven were later given roentgen treatment without any perceptible effect on the disease. Five had been treated with roentgen radiation before treatment with phosphorus, with partial control in 2 and no control in 3. Five of these cases were classified as leukosarcoma which is an acute form of lymphosarcoma.

The remissions after phosphorus treatment seem to be longer than after roentgen treatment.

For phosphorus to attain its maximum effectiveness the lesions must be reasonably radiosensitive. Because it reaches all areas of the disease, phosphorus is not only a good method of primary therapy but a valuable adjunct to roentgen therapy in many cases. If it were given routinely to all patients who have responded to roentgen irradiation with good regression of symptoms further inactivation might take place in those areas and hidden foci might also be inactivated. This should prolong the periods of remission and might in some cases prolong the periods of remission and might in some cases completely cure the disease.

It is believed that phosphorus should be used either primarily or secondarily in practically all cases of lymphosarcoma.—*Audrey G. Morgan.*

FITZ-HUGH, THOMAS, JR., and HODES, PHILIP J. Clinical experience with radiophosphorus in the treatment of certain blood dyscrasias. *Am. J. M. Sc.*, Nov., 1942, *204*, 662–665.

The authors have been supplied with enough radiophosphorus to treat 38 patients, 8 of them with polycythemia vera, 5 with chronic myelogenous leukemia, 4 with chronic lymphatic leukemia, 4 with acute leukemia, 5 with Hodgkin's disease, 6 with lymphosarcoma, 1 with multiple myeloma, 1 with reticulo-endotheliosis, 1 with reticulum cell sarcoma and 3 with metastatic carcinoma.

Radiophosphorus has an atomic weight of 32 and is therefore called P^{32}. It is produced by bombarding ordinary red phosphorus whose atomic weight is 31 with rapidly moving deuterons in a cyclotron. It has all the properties of ordinary phosphorus with the addition of radioactivity.

The material in solution may be given by mouth or injection. Oral doses vary from 1 to 20 mc. and intravenous doses from 0.2 to 2 mc. Total dosage varies with the hematologic and clinical condition of the patient and his response to treatment; also the frequency of dosage. Some patients are given a dose once a week, some 3 or more times a week.

Radiophosphorus has proved at least equal in value to other methods of radiation therapy in some of the malignant blood dyscrasias. It has some advantages, such as ease of administration, absence of marked radiation sickness, more concentrated effect on bone marrow and more prolonged remissions in chronic leukemia and polycythemia vera. The authors have not found it of any value in the acute leukemias.—*Audrey G. Morgan.*

MARINELLI, LEONIDAS D. and GOLDSCHMIDT, BERTRAND. The concentration of P^{32} in some superficial tissues of living patients. *Radiology*, Oct., 1942, *39*, 454–463.

This paper describes the technique used in determining the concentration of P^{32} in normal and pathological skin and gives the preliminary results found in 3 patients who were taking treatment with radioactive phosphorus. One of these patients had melanoma, the other two mycosis fungoides. The P^{32} concentration was determined by means of a Geiger-Müller counter.

It was found that the activity was greater in skin overlying bone than in that overlying soft tissues. In all 3 pathological cases the rate of uptake and rate of elimination of the P^{32} were radically different from those in normal skin. In the case of melanoma in the course of time it was found that the activity of the melanotic nodules increased in proportion to that of normal skin. On comparing the P^{32} turnover in Cases 2 and 3 of mycosis fungoides it was found that it was greater in Case 2 than in Case 3; the former case was the more malignant. This is in accordance with the rule that radioactive phosphorus is metabolized in greater amounts by malignant that by normal tissue. This suggests the possibility of using the turnover of radioactive elements as a quantitative index of the degree of malignancy of tumors. Needless to say, the possession of such an index of malignancy would be of great value in the study of tumors.—*Audrey G. Morgan.*

RASMUSSEN, RICHARD A., and ADAMS, W. E. Experimental production of emphysema. *Arch. Int. Med.*, Sept., 1942, *70*, 379–395.

Experiments were made on dogs in an effort to determine the cause of emphysema. The dogs were subjected to intermittent overinflation of the lungs simulating repeated attacks of asthma over long periods. Details of the experiments are given and photomicrographs of the histological findings.

Biweekly pulmonary overinflation alone with a pressure of 35 mm. mercury over periods as long as eleven months failed to produce true emphysema in dogs. The pressures used were high enough to cause marked lowering of the systemic arterial pressure and a marked rise of the peripheral venous pressure, and in 2 cases pulmonary air embolism and hemorrhage into the lung parenchyma.

Emphysematous changes were brought about in the lungs of one dog which showed evidence of an old pneumonia. This suggests that a combination of infection and overdistention may be the cause of emphysema.—*Audrey G. Morgan.*

THE AMERICAN JOURNAL OF ROENTGENOLOGY AND RADIUM THERAPY

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GENERAL INFORMATION

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THE AMERICAN JOURNAL OF ROENTGENOLOGY AND RADIUM THERAPY

VOL. 51 MAY, 1944 NO. 5

THE DIAGNOSIS OF BRONCHIECTASIS IN YOUNG ADULTS

PREBRONCHOGRAPHIC ROENTGEN MANIFESTATIONS OBSERVED AMONG MILITARY PERSONNEL

By CAPTAIN WILLIAM A. EVANS, JR.
Medical Corps, Army of the United States
and
CAPTAIN LEON J. GALINSKY
Medical Corps, Army of the United States

BRONCHIECTASIS has been the most common chronic pulmonary condition at this Station Hospital. This has also been the experience of Thomas.[1] The high incidence during a period of a few months in a group of apparently healthy young men who had recently passed draft board examinations was surprising to us. Pulmonary tuberculosis appears to have been excluded far more effectively at induction stations. While tuberculosis is undoubtedly of great epidemiological importance, bronchiectasis carries with it a prognosis as serious for the life and health of the individual. From both military and industrial standpoints, with their similar responsibilities for disability compensation, bronchiectasis—its course characterized by repeated severe respiratory tract infections and culminating in early death—assumes a significance of more than academic interest.

Inasmuch as the recognition of bronchiectasis depends upon a critical estimate of the patient's history, clinical manifestations and roentgenographic observations, it seemed profitable to review our series particularly in regard to the prebronchographic roentgen manifestations in the lungs. For various reasons, the soldier's history as revealed by response to questioning proved in general to be unreliable and the roentgen data therefore assumed a more prominent rôle in raising the suspicion of bronchiectasis. Diamond and Van Loon[2] have similarly noted that "the clinical picture is not a reliable index of the degree of anatomic change." By and large, the patients in this series were admitted to the hospital with acute respiratory infections and were under a period of observation for some weeks or months before bronchograms were made. In the study of the roentgenograms obtained during this period, certain prebronchographic signs of bronchiectasis became evident. The roentgenograms were then reviewed to determine the reliability of these signs in the diagnosis of bronchiectasis.

The material consisted of 95 cases in which bronchograms had been made and where prebronchographic roentgenograms were available for examination. Frank

bronchiectasis was observed in 37 cases, minimal or questionable bronchiectasis in 24 and no bronchiectasis in 34. Of the 37 cases with frank bronchiectasis, the location was observed to be in the right lung in 13, in the left lung in 16 and bilateral in 8. A similar distribution was obtained with the minimal or questionable bronchiectasis included (Table 1).

TABLE I

LOCATION OF BRONCHIECTASIS

	Bronchiectasis		
	Minimal	Frank	Total
Right lung	13	13	26
Left lung	9	16	25
Both lungs	2	8	10
No bronchiectasis	—	—	34
Total	24	37	95

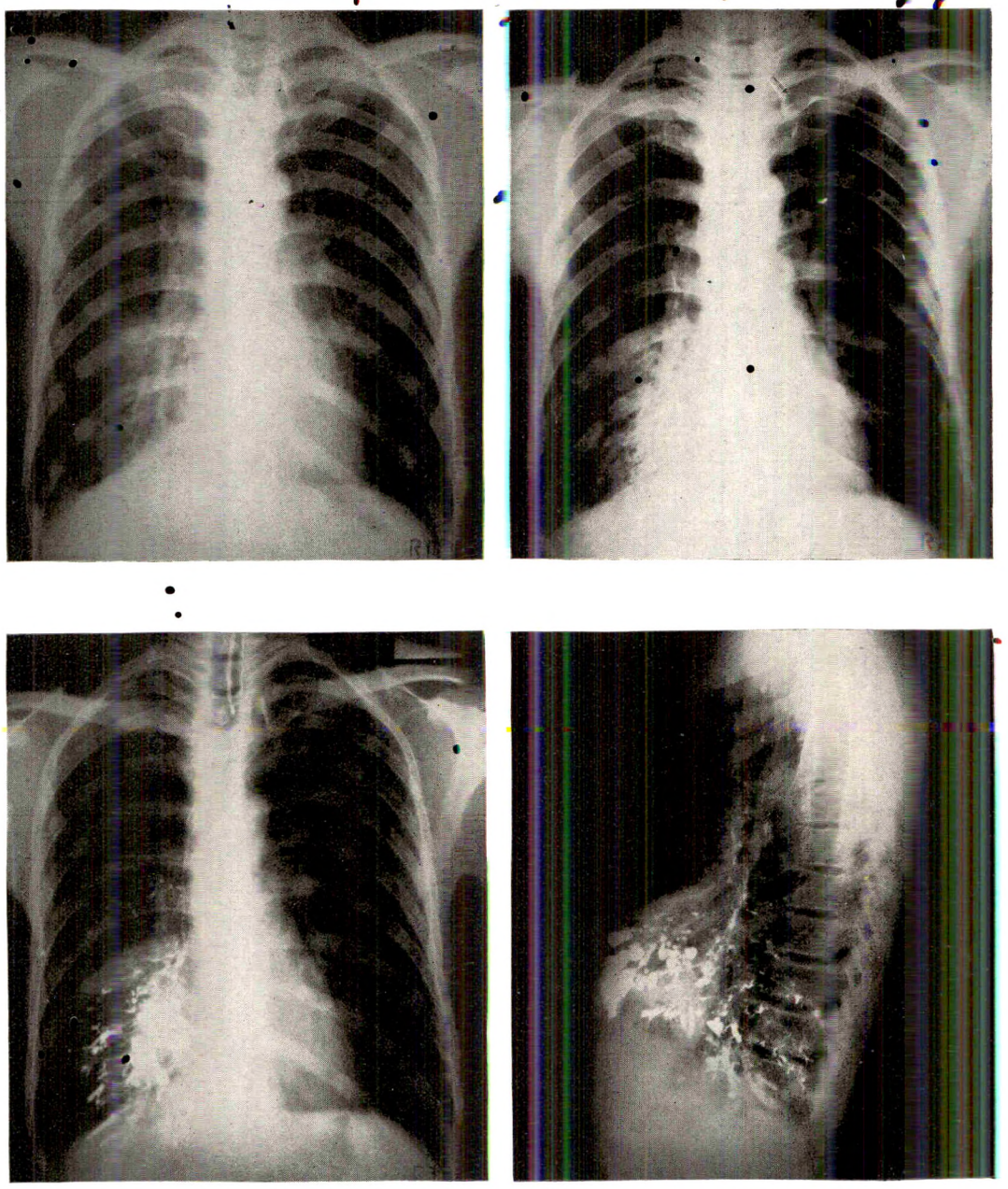
PREBRONCHOGRAPHIC ROENTGEN EVIDENCE OF BRONCHIECTASIS

Slowly Resolving Bronchopneumonia (Fig. 1*A* and 1*B*). The majority of the patients showed evidence of a basal bronchopneumonic infiltration on the initial roentgenogram. Often the symptoms were mild and the appearance of the lesion would have warranted the diagnosis of "primary atypical pneumonia" now acceptable in many quarters. Usually these small basal infiltrations resolve completely in a period of ten to fourteen days. When the lesion shows only partial resolution in two weeks and persists for three or four weeks or longer, bronchiectasis should be strongly considered. In this series a slowly resolving bronchopneumonia was observed in 28 of the 37 cases with frank bronchiectasis, in 14 of the 24 with minimal or questionable bronchiectasis and in 11 of the 34 where no bronchographic evidence of bronchiectasis was obtained.

Prominent Pulmonary Markings (Fig. 2). A frequent but less definite and reliable sign of bronchiectasis was the accentuation of the bronchial markings in the basal and peripheral lung fields. This was interpreted as a peribronchial infiltration. Normally the bronchial markings become very fine and inconspicuous as they approach the periphery of the lung but when they are accentuated just above the diaphragm and especially in the costophrenic sulcus, bronchiectasis may be suspected. The extent and character of the hilar and perihilar deposits do not seem to be of so much importance in this regard. A basal peribronchial infiltration is frequently observed in association with a slowly resolving bronchopneumonia. Prominent pulmonary markings of this character were observed in 29 of the 37 cases with frank bronchiectasis, in 13 of the 24 with minimal or questionable bronchiectasis and in 20 of the 34 with no bronchographic evidence of bronchiectasis.

Recurrent Bronchopneumonia (Fig. 3). A less common but nevertheless dependable sign of bronchiectasis was the observation of a pneumonia recurring in the same area of the lower lung fields after an interval of a few weeks or months. Undoubtedly if the observations had extended over a period of years, this would have been a more frequent and reliable indication of bronchiectasis. In our series a recurrent bronchopneumonia was found in 6 of the 37 cases with a frank bronchiectasis, and in 2 cases where no bronchiectasis was detected bronchographically.

Atelectasis (Fig. 4). For a number of years the association of bronchiectasis and atelectasis has been remarked. The recognition of a triangular segmental area of atelectasis in the lower medial lung fields, particularly in the course of pneumonia in children, has been described as a forerunner or accompaniment of bronchiectasis. The relationship of atelectasis and bronchiectasis has been examined experimentally by Tannenberg and Pinner.[3] They conclude: "A comparison of the experimental observations with the changes seen in human pathology makes it more probable that uncomplicated atelectasis per se has no bearing whatever on the development of bronchiectasis.



5.12.43 6.12.43

FIG. 1*A*. Basal bronchopneumonia persisting with very little change during a period of eighteen days, observed in a patient examined routinely for an over-age discharge. Inquiry elicited a history of mild, chronic productive cough following pneumonia in 1925. Bronchograms, made on the basis of roentgen interpretation, revealed an advanced saccular bronchiectasis with some atelectasis involving particularly the right middle lobe.

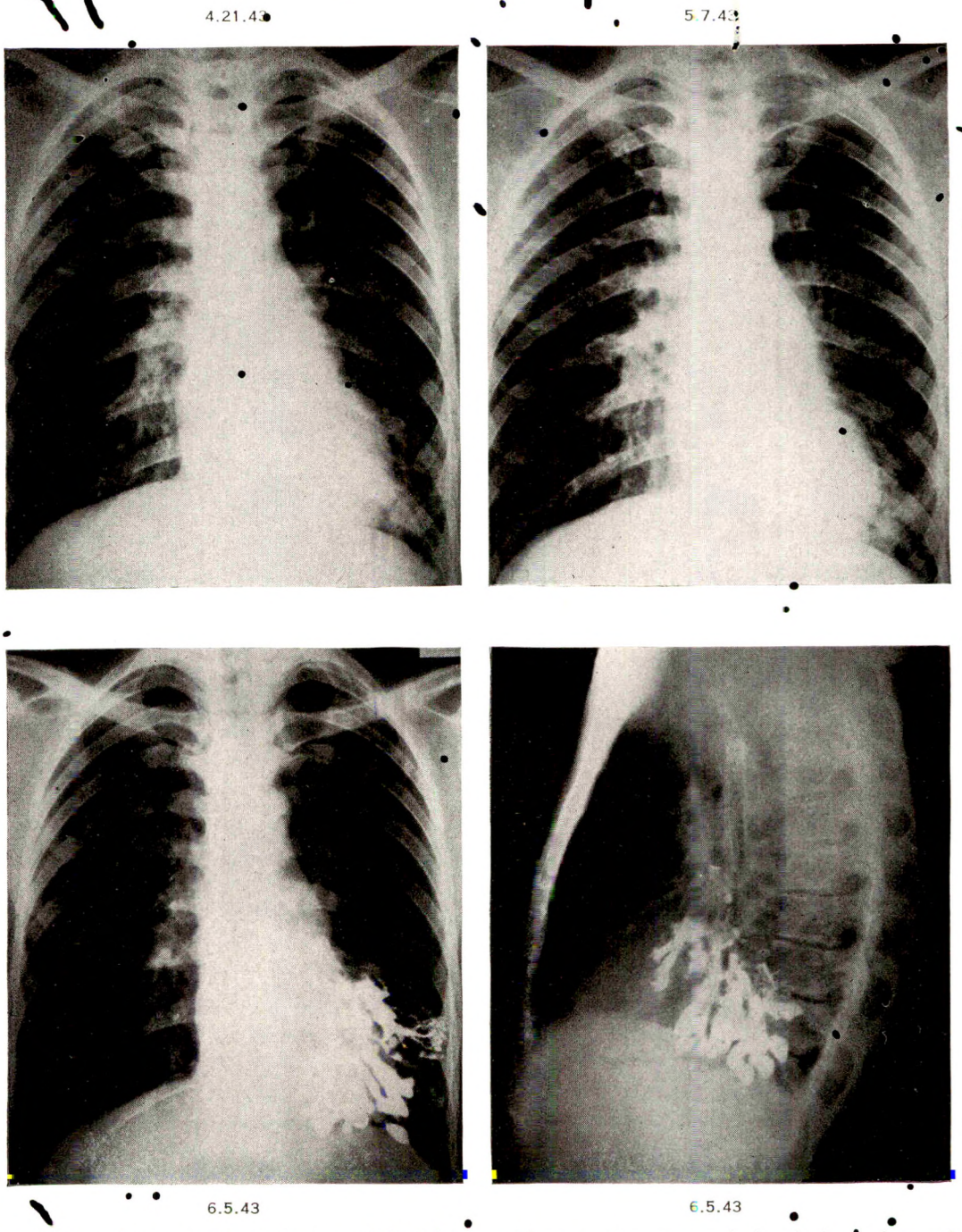
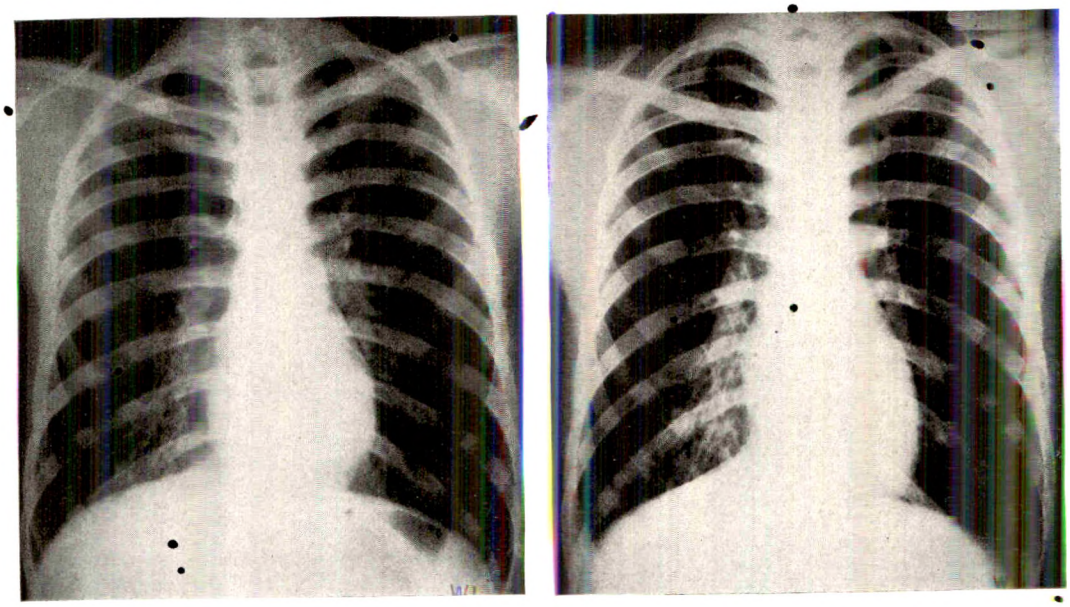


FIG. 1B. Bronchopneumonia at the base of the left lung without tendency to resolution during a sixteen day observation period in a patient admitted to the hospital with clinical evidence of acute respiratory infection. History disclosed three attacks of pneumonia in childhood, with occasional but mild subsequent respiratory infections. An advanced saccular bronchiectasis was demonstrated in the left lower lobe in bronchograms made on recommendation of the roentgenologist.

3.27.43 6.11.43



6.19.43 6.19.43

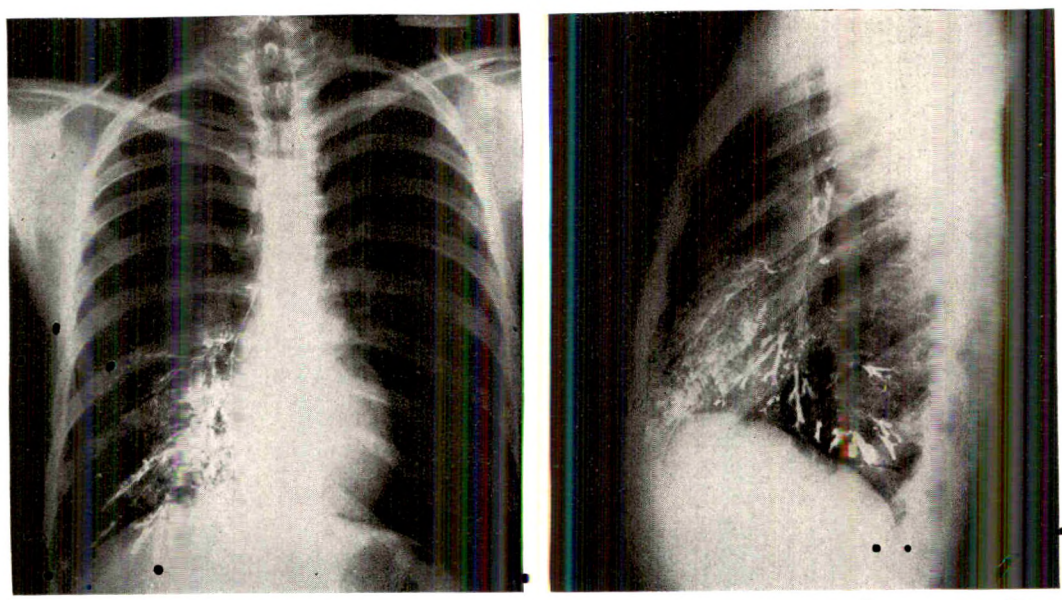


FIG. 2. Prominent bronchial markings observed in the lower medial portion of the right lung over a period of two and one-half months in a patient with no complaint but with a history of frequent respiratory tract infections associated with protracted moderately productive cough. Bronchograms made at the suggestion of the roentgenologist demonstrated a moderate cylindrical bronchiectasis at the base of the right lower lobe.

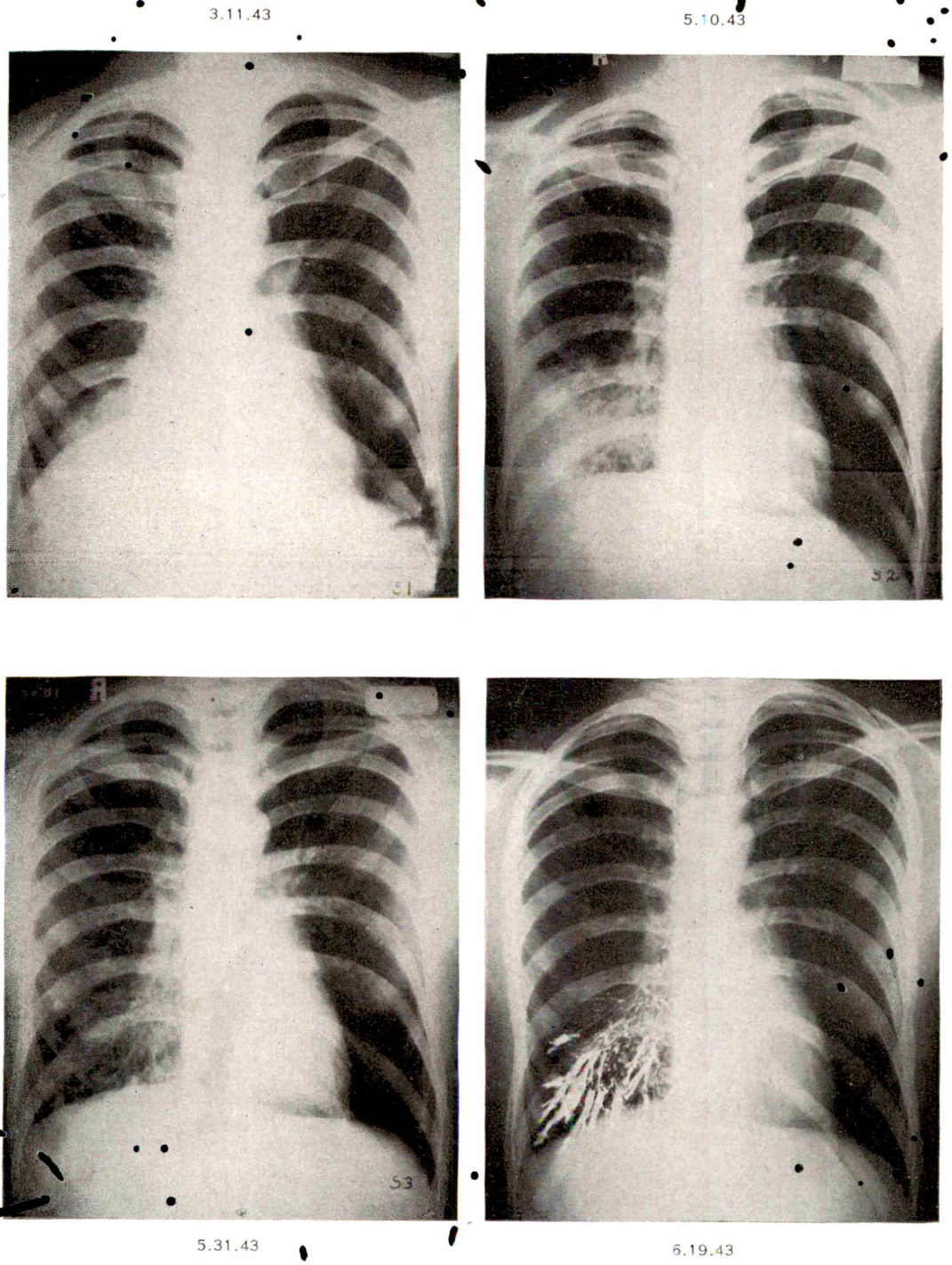


FIG. 3. Recurrent basal bronchopneumonia within an interval of two months and evidence of slow resolution with persistent peribronchial infiltration in the second attack. The patient gave a history of a severe pneumonia in childhood, but subsequent respiratory tract infections were infrequent and mild. The prebronchographic roentgenograms were considered strongly indicative of bronchiectasis and bronchograms demonstrated a cylindrical right lower lobe bronchectasis.

There is, however, another relationship of these two conditions. Both may be produced by the same cause. Atelectasis in children and the other primary diseases which are found frequently in the history of bronchiectasis are caused almost without exception by inflammatory conditions in the lung. Bronchial exudate and inflammatory swelling of the bronchial mucosa may close the bronchial lumen and cause atelectasis within a short time." A sharply defined segmental area of complete atelectasis was not observed in this series but we were impressed by the occasional occurrence of a shrunken, contracted area of lung in the basal or lower medial lung fields. This was sometimes manifested by some degree of contraction of the thoracic cage as compared with the opposite side, but more often by a compression and retraction of the bronchial markings toward the base and mediastinum. The observation of this abnormal configuration of the bronchial tree was then confirmed bronchographically by the demonstration of the dilated bronchial radicles closely packed against the mediastinum or the dome of the diaphragm. This type of atelectasis would seem to be the result of a fibrosis in the involved pulmonary segment rather than the mechanical effect of simple bronchial obstruction. The prebronchographic evidence of a contracted lung was observed in 11 cases of the 37 with frank bronchiectasis, in 5 of the 24 with minimal or questionable bronchiectasis, and 3 of the 34 where no bronchiectasis was proved.

These data are summarized in Table II.

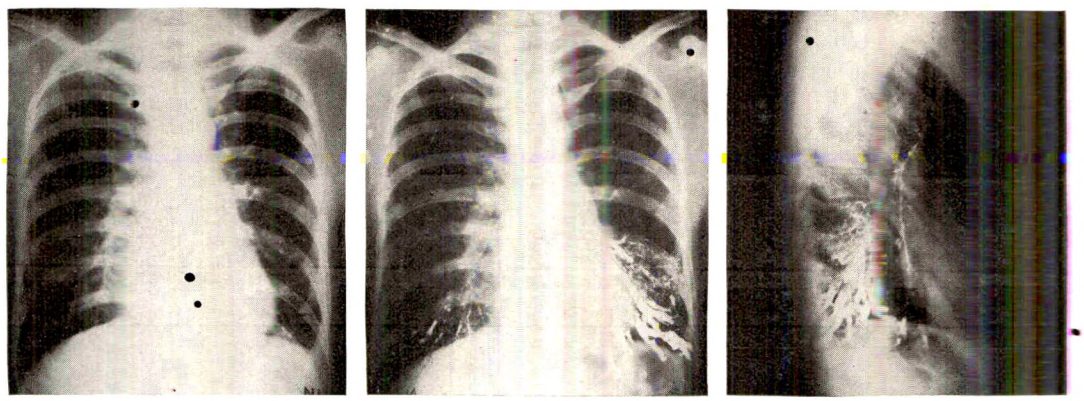


FIG. 4. Prebronchographic roentgenogram in a patient with an intermittent mild productive cough associated with common respiratory infections shows a contraction of the left lower chest and compression of the pulmonary markings at the base of the left lung toward the diaphragm and mediastinum. There was a history of pneumonia complicated by empyema on the left in 1937. Bronchograms show an advanced cylindrical and saccular bronchiectasis with a high degree of contraction of the left lower lobe, as revealed by the close proximity of the bronchial radicles.

BRONCHOGRAPHIC EVIDENCE OF BRONCHIECTASIS

In this study lipiodol was introduced into the bronchial tree by an oral cannula under indirect vision after cocainization of the oropharynx. The lipiodol was directed toward the part of the bronchial tree under suspicion by positioning the patient. The bronchographic demonstration of cylindrical and saccular dilatations of the bronchi to establish the diagnosis of bronchiectasis is generally accepted and requires no comment. Of particular interest to us was the detection of lesser changes in the group of patients classified as having minimal or questionable bronchiectasis. It is our assumption that no sharp distinction can be made between the normal and the ectatic

bronchus but that imperceptible gradations exist in the deviation from normal. It is not surprising, therefore, in consideration of minor variations of the normal pattern, that difficulty is encountered in determining what constitutes an abnormal bronchial tree and what evaluation should be assigned to these variations. What significance should be attached to the finding of one or two small saccular dilatations in the bronchial tree or to a slight irregularity in the contour of the lumen of a few small bronchial radicles or to an absence of the gradual diminution in caliber normally observed as the bronchus approaches the periphery of the lung?

The evaluation of such bronchographic signs depends upon our concept of bronchiectasis and this is subject to further enlightenment. Is bronchiectasis invariably progressive with more advanced involvement and the likelihood of a fatal termination if the abnormal lung is not removed, or may these minor bronchial abnormalities be self-limited, either being arrested in a harmless state or even regressing toward a normal pattern? The observation of these patients with minor bronchial abnormalities over a period of years may do much to elucidate our ideas on the pathogenesis and development of advanced bronchiectasis.

It was the observation of Diamond and Van Loon[2] that "Spread of the bronchiectatic process to previously uninvolved bronchi was not noted in any of the patients. The size of the existing dilatations remained unchanged in all but three. Reference has been made by Anspach and other investigators to the tendency of dilatations, when once formed, to increase in size with the passage of time. Whatever the tendency toward progression may be in adult life, it is clearly evident from the data in our series that such a tendency is not a prominent feature of bronchiectasis during childhood." These opposing views of Anspach and Diamond and Van Loon illustrate the difficulty in determining the "line of duty" status of bronchiectasis in the Army. It is our conception, in the light of the frequent history of childhood pneumonia, and of such studies as those of Diamond and Van Loon, that the bronchiectasis antedates the recent pulmonary infection, and probably has its origin in childhood. The rôle of whooping cough and pneumonia during childhood in the production of bronchiectasis is well recognized. Subsequent pneumonic infections may depend for their localization upon the presence of an underlying bronchiectasis. These infections represent therefore an incident in and a manifestation of the course of the disease

TABLE II

INCIDENCE OF PREBRONCHOGRAPHIC SIGNS

Sign	Bronchiectasis					
	None		Minimal		Frank	
	Most prominent sign only	Additional signs included	Most prominent sign only	Additional signs included	Most prominent sign only	Additional signs included
No sign	3	—	2	—	1	—
Slowly resolving pneumonia	17	11	12	14	20	28
Prominent pulmonary markings	13	20	10	13	8	29
Recurrent pneumonia	1	2	—	—	6	6
Contracted lung	0	3	0	5	2	11
Total	34		24		37	

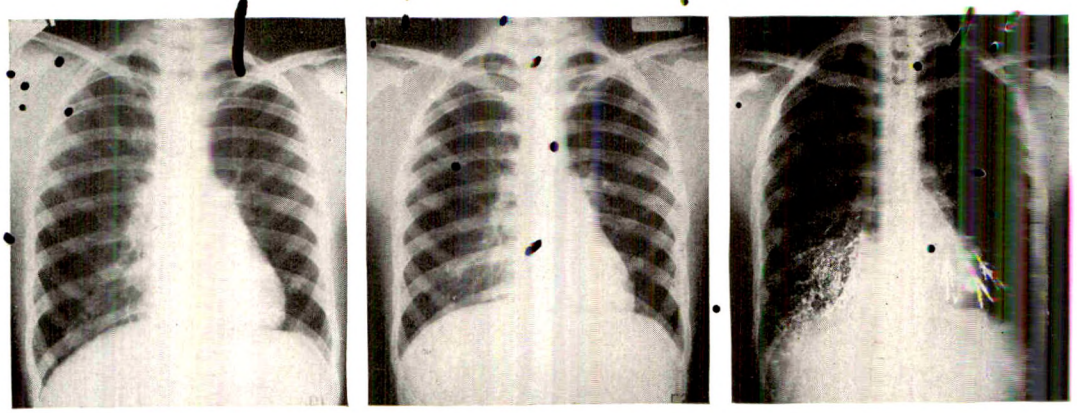


FIG. 5. Roentgenograms of the chest made during hospitalization for mild upper respiratory infection disclosed an abnormal appearance of the left lower lung field with a deficiency of the normal bronchial markings, suggesting a congenital cystic malformation. This was also observed at a subsequent examination five weeks later and bronchographic studies were suggested. Bronchograms revealed a bronchiectasis in the left lower lobe behind the heart, with a contraction of this portion of the lung and a compensatory emphysema of the adjacent lung. No characteristic symptoms or signs of this condition were elicited.

rather than a factor in its production and development.

CORRELATION OF THE PREBRONCHOGRAPHIC AND BRONCHOGRAPHIC ROENTGEN SIGNS

An analysis of the tabulated results (Table III) revealed that bronchiectasis as demonstrated by bronchography could be predicted with reasonable accuracy from a study of the prebronchographic roentgenograms. A fairly close correlation was obtained in 32 cases with frank bronchiectasis, in 21 with minimal or questionable bronchiectasis, and in 26 where no bronchiectasis was discovered. A poor correlation was found in 5 cases having a frank bronchiectasis, in 3 with minimal or questionable bronchiectasis, and in 8 with no bronchographic evidence of bronchiectasis. There was therefore a tendency to suspect bronchiectasis somewhat more frequently than confirmation could be obtained bronchographically. In some instances, a more accurate prebronchographic diagnosis could probably have been achieved if there had been a longer preliminary observation with serial roentgenograms. Two cases are illustrative of positive and negative errors that were made. In one (Fig. 5) bronchiectasis was discovered in the bronchographic study of what was thought to be a congenital malformation of the lung. The malformed lung proved to be an emphysema secondary to a contracted bronchiectatic area of lung behind the heart. In the other (Fig. 6) there seemed to be adequate evidence to warrant a diagnosis of bronchiectasis which could not, however, be confirmed bronchographically. It thus seems to us advisable to seek bronchographic confirmation of a diagnosis of bronchiectasis, however strong the suspicion for such a diagnosis may have been from previous clinical roentgen studies.

TABLE III

CORRELATION OF PREBRONCHOGRAPHIC SIGNS WITH BRONCHOGRAPHIC EVIDENCE OF BRONCHIECTASIS

	Bronchiectasis			
	None	Minimal	Frank	Total
Good correlation	26	21	32	79
Poor correlation	8	3	5	16
Total	34	24	37	95

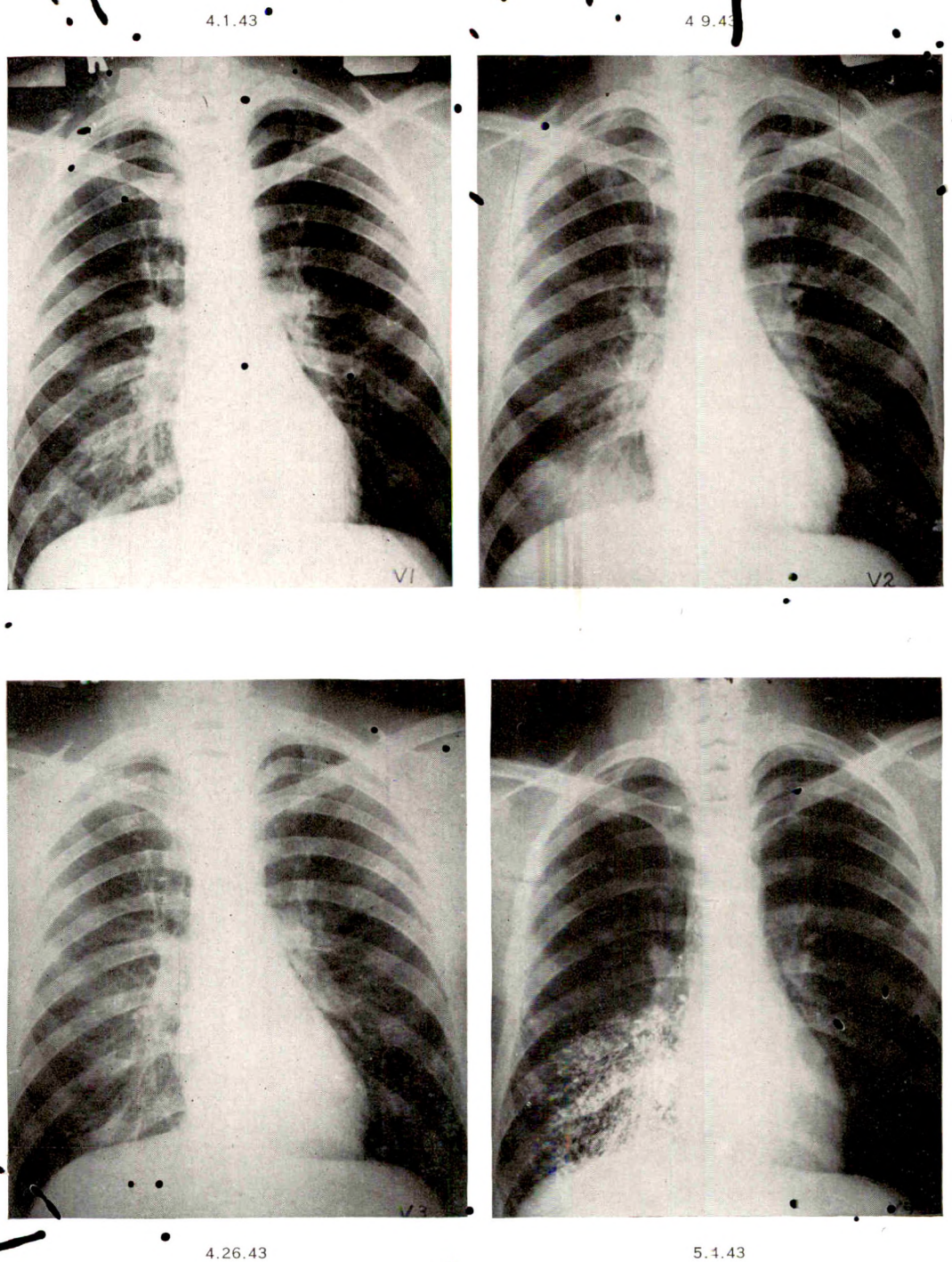


FIG. 6. A low grade, slowly resolving basal bronchopneumonia in the right lung observed in a patient with no significant antecedent respiratory history. The slow resolution was accompanied by a persistent peribronchial infiltration and some contraction of the involved lung. No bronchiectasis was demonstrable.

CONCLUSIONS

1. The more frequent recognition of bronchiectasis derives from an awareness on the part of the clinician and roentgenologist that the condition may exist in the absence of the classical signs and symptoms.

2. The observation in serial roentgenograms of a slowly resolving basal bronchopneumonia, recurrent basal bronchopneumonia, persistent basal peribronchial infiltration, and a contracted segment of lung, either singly or in combination, warrants a suspicion of bronchiectasis.

3. With serial roentgen studies during a period of several months a reasonably accurate diagnosis of bronchiectasis may be made, but

4. Bronchographic studies are necessary to confirm the diagnosis.

5. Observation of patients with minimal bronchial changes during a period of years is desirable to learn the significance of these changes.

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3. TANNENBERG, J., and PINNEE, M. Atelectasis and bronchiectasis. *J. Thoracic Surg.*, 1942, *11*, 571–616.

ORNITHOTIC PNEUMONIA*

By A. MELAMED, M.D., *and* JACOB M. FINE, M.D.†
MILWAUKEE, WISCONSIN

IT IS becoming increasingly apparent from the recent reports of Meyer,[4] Meyer and Eddie,[5] Meyer, Eddie and Yanamura,[6] Smadel,[7] Favour[3] and others[1,2] that many of the so-called cases of atypical or virus pneumonia are due to a psittacine or psittacine-like virus. Smadel[7] found at least 10 of 45 sporadic cases of atypical pneumonia in large eastern urban populations to be due to infections with strains of the virus of psittacosis. Favour[3] makes some interesting speculations:

> If it is true that bacteria-free atypical pneumonia other than ornithosis was not known before 10 years ago, one of the ornithosis viruses might be offered as the progenitor of human atypical pneumonia, having become so established as a result of the 1929–30 psittacosis epidemic. This presumes a very rapid adaptation in man. It is more likely, however, that atypical (virus) pneumonia is an ancient disease. As in endemic Australian ornithosis, perhaps some strains of the virus of human atypical pneumonia are well adapted to homogeneous human infection chains, while other strains, like the South American ornithosis virus, still require heterogeneous hosts to insure their perpetuation, and therefore may be passed to experimental animals.

The value of the work and reports of Meyer and others on the subject of ornithosis cannot be overestimated. Were it not for such recent reports our examples of ornithosis would probably have been considered as cases of primary atypical pneumonia, etiology unknown. Awareness of the possibility of such an infection led to further questioning of the patients, which in at least 2 of our 3 cases revealed pertinent facts, and investigation of the patients' sera for ornithosis.

CASE REPORTS

CASE 1.* B. L. (110203), male, aged sixty-six, entered the hospital on March 23, 1941, soon after having returned to Milwaukee from a vacation in Florida and Georgia where he visited with many friends who were engaged in farming occupations. The patient stated that his illness dated back to January, 1941, but four weeks previous to entering the hospital he had the "flu" with severe sore throat and enlarged glands in the neck. Soon after the "flu" had subsided his knees, ankles and left elbow became swollen, stiff, painful and slightly reddened. Massage and heat therapy offered only slight relief.

The patient was a retired cattle dealer, having retired a few years before the onset of this illness.

Examination. The patient did not appear acutely ill when he entered the hospital; he lay quietly in bed. His blood pressure was 110/70, temperature 101.1° F., pulse 100, respirations 22. Physical examination of the chest was essentially negative. Both knees, both ankles and the left elbow were swollen, tender, reddened, and showed limitation of motion. The clinical impression was acute rheumatic fever.

Laboratory Data. The red blood cell count on admission was 3,810,000 per cu. mm., hemoglobin 12 grams, leukocytes 31,300 per cu. mm. with 95 per cent polymorphonuclear leukocytes. The red blood cells showed stippling, slight toxic degeneration, slight polychromatophilia, poikilocytosis, anisocytosis, and hypochromia. The sedimentation rate of the red blood cells was 48 per cent in fifteen minutes, 40 per cent in thirty minutes, 37 per cent in sixty minutes. The blood Kline reaction was negative. Agglutination tests for typhoid and paratyphoid fever and undulant fever were also negative. Blood cultures were sterile seven days after inoculation

* Referred by Dr. C. E. Bellehumeur.

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A roentgenogram of the chest taken April 7, 1941 (Fig. 1), approximately two weeks after admittance to the hospital or six weeks after the attack of "flu," showed a diffuse veil-like shadow overlying the entire left chest. The underlying lung detail was not clearly discernible. The left diaphragm and costophrenic space were obscured. A few suggestive infiltrations were seen in the base of the right lung. The roentgen appearance of the chest was indicative of a diffuse pleural reaction on the left side with a possible underlying pneumonic process. Suggestive areas of bronchopneumonia were also present in the right base.

Course. With the exception of slight elevation during the first two days in the hospital, the patient's temperature was normal for a period of approximately one week. It then began to rise steadily until it reached 105° F. The temperature was elevated for two weeks and then fell rather sharply to 100° F. and slowly returned to normal after a few days.

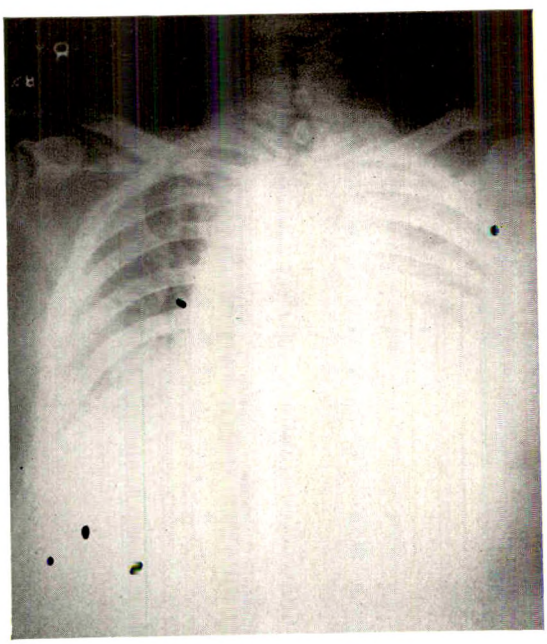


FIG. 1. Case 1. Portable chest roentgenogram taken April 7, 1941. Obscuration of entire left chest by large pleural effusion. Infiltrations in right base.

Electrocardiographic tracings made on May 29, 1941, showed a tendency to right axis deviation with merely suggestive evidence of myocardial involvement. The tracings were not repeated.

Barium enema examination was performed April 14, 1941, after the patient developed some abdominal distention. There was no evidence of an organic lesion in the colon.

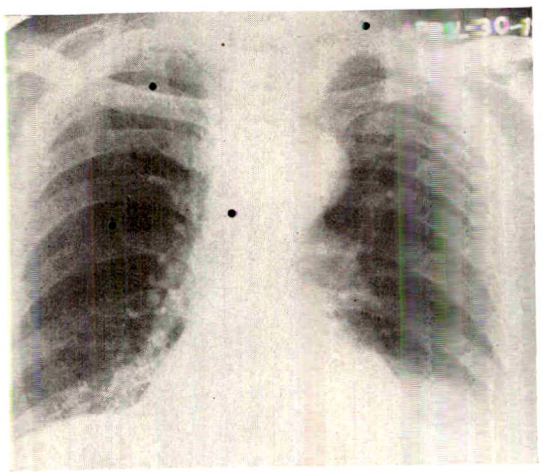


FIG. 2. Case 1. Roentgenogram made April 30, 1941, approximately five weeks after admission to hospital. Slight pleural reaction still present in left lower lateral chest region.

Sulfadiazine administration showed no apparent effect on the course of the disease. Supportive and symptomatic treatment including a blood transfusion was administered. The high white blood cell count and polymorphonuclear leukocytosis persisted throughout the hospital confinement.

Subsequent chest roentgenograms taken April 15, 21 and 30, 1941 (Fig. 2) showed gradual resolution of the pneumonic and pleural processes, the latter on the left side.

The patient was discharged from the hospital in apparently good condition on May 1, 1941, forty days after admission or approximately nine weeks after the onset of "flu."

Follow-up Record. During the month of November, 1941, the patient complained of joint pains and weakness. Although the patient had no knowledge of contact with infected birds, Dr. J. S. Goodman submitted a sample of blood serum to Dr. K. F. Meyer of George Williams Hooper Foundation, University of California, San Francisco. Dr. Meyer promptly reported a 4 plus complement fixation reaction

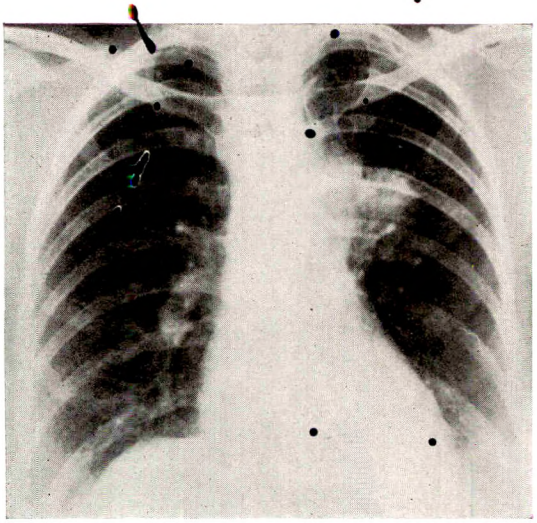


FIG. 3. Case II. Roentgenogram taken May 23, 1942, approximately eight days after onset of illness, shows a circumhilar shadow on left side.

with psittacine antigen in a dilution of 1:32. The reaction was checked by tests with a second specimen of blood serum and Dr. Meyer remarked that there was no doubt in his mind that the patient suffered from ornithosis and that the original infection probably occurred during the early part of the year 1941.

As far a can be determined at the present time the patient is in good health and fully recovered from this illness.

CASE II. *Mrs. R. R. (118060), aged fifty, was admitted to Mount Sinai Hospital on May 23, 1942. One week before admission the patient contracted a "cold" and began to complain of malaise, joint pains and dull pain in the lower abdomen. On May 20, 1942, the visiting physician made a diagnosis of early pneumonia.

The past history as obtained from the patient on admission to the hospital was essentially negative except for a history of chronic gallbladder disease.

Examination. Physical examination revealed an elderly, fairly well nourished, white female, rather gravely ill and showing a slight degree of cyanosis. Her skin was hot and dry. Some pharyngitis was present. Physical examination of the chest was essentially negative. Abdominal palpation revealed some tenderness in the left lower abdomen and evidence of splenic enlargement. The blood pressure was 120/66, temperature 101.2° F., pulse 94, respiration rate 20. The clinical impression on admission was central pneumonia or septicemia.

Laboratory Data. Blood determinations showed the following: Hemoglobin 12.5 grams, red blood cells 3,940,000 per cu. mm., leukocytes 7,600 per cu. mm., polymorphonuclears 75 per cent, lymphocytes 18 per cent, basophiles 1 per cent, stippled red blood cells, polychromatophilia, toxic granulation and slight anisocytosis. Urinalysis showed a slight trace of albumin and a fairly large number of leukocytes (25 or 30) per high power field. The sedimentation rate of the red blood cells was 53 per cent in fifteen minutes, 43 per cent in thirty minutes and 38 per cent in one hour. The blood Kline test was negative.

A roentgenogram (Fig. 3) of the chest made May 23, 1942, the day of admission to the hospital, showed a circumhilar area of increased density measuring approximately 6 cm. in diameter on the left side. No other definite abnormality was noted in the lungs. Roentgenologically, one was unable to differentiate between central or hilar pneumonia, lung abscess or even lung neoplasm.

Course. The patient's condition became progressively worse during the following two weeks. The temperature ranged between 100.0 and 105.8° F., rectally. Occasionally the patient complained of chilly sensations. Blood culture

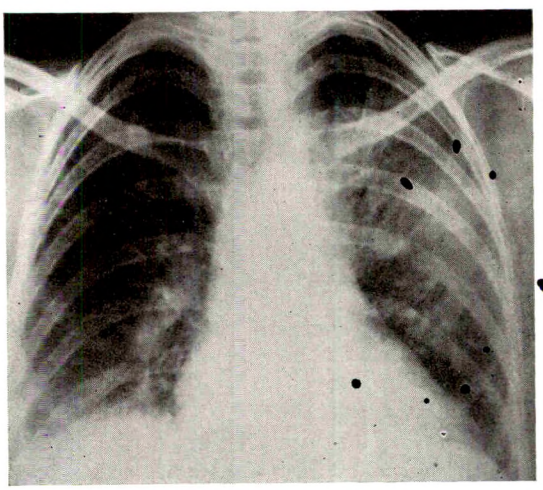


FIG. 4. Case II. Fan-shaped involvement of left upper pulmonary lobe three days following first roentgen examination.

* Referred by Dr. N. A. Gendlin.

showed no growth seven days after inoculation. Throat culture showed predominance of *Streptococcus viridans*. Gastric washings were negative for acid-fast bacilli. Culture of a small amount of material obtained by thoracentesis was sterile seventy-two hours after inoculation.

Roentgen examination of the chest on May 26, 1942 (Fig. 4), approximately ten days after the onset of the disease, showed more extensive and fan-shaped involvement of the left upper lobe.

A roentgenogram of the chest on June 4, 1942 (Fig. 5), approximately two weeks after the original examination or three weeks after the onset of illness, showed almost complete resolution of the pathologic condition previously described in the left subclavicular region, but definite evidence of the pneumonic process was noted extending downward and outward from the hilar zone in the right lung. Some peripheral clouding of the left base was also noted. The roentgen appearance of the lungs was consistent with a diagnosis of atypical (virus) pneumonia, etiology unknown.

The patient showed no apparent satisfactory or favorable response to any of the therapeutic measures employed. Sulfadiazine, sulfanilamide and sulfathiazole were administered on various occasions in doses of 15 grains every four hours. Streptococcus convalescent serum showed no effect. Three deep roentgen treatments were

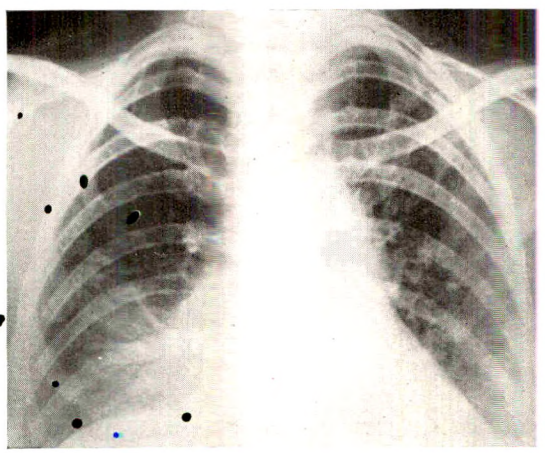


FIG. 5. Case II. Roentgen appearance of chest on June 4, 1942, three weeks after onset of disease. Almost complete resolution of left upper lobe pneumonic process. New infiltrations extending downward and outward from lower right hilar region.

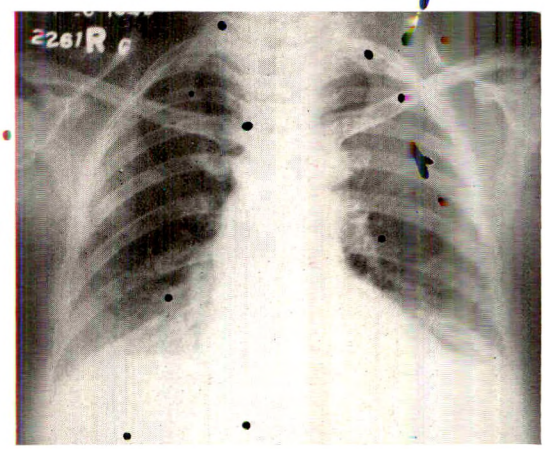


FIG. 6. Case III. Roentgenogram of chest (May 20, 1943) shows faint bronchopneumonic areas in the right base.

given to the left chest and one treatment was given to the right chest without any effect on the clinical course. Several blood and plasma transfusions were given. The white blood cell count never rose above 8,600 per cu. mm.

Agglutination tests for typhoid and paratyphoid fever and brucellosis abortus were negative. Further questioning of the patient, approximately two weeks after the patient was admitted, revealed that the patient had handled and eaten pigeons two weeks before entering the hospital. Blood serum was then immediately sent to Dr. K. F. Meyer, George Williams Hooper Foundation, for complement fixation tests for psittacosis. Dr. Meyer reported a 4 plus reaction in dilution of 1:64 and a 3 plus reaction in a dilution of 1:128. The serum, in Dr. Meyer's opinion, was strongly positive for psittacosis.

The patient began to improve after three weeks of hospitalization and a subsequent roentgen examination of the chest on June 19, 1942, showed very few residual pulmonary infiltrations in either lung. The patient was discharged from the hospital on July 4, 1942, in good condition. The duration of this patient's illness was approximately six weeks.

CASE III. *Mrs. M. F. (126983), aged fifty-three, entered the hospital on May 16, 1943, complaining of fever and left frontal headaches of one week duration. The patient stated that the fever ranged between 101 and 103° F.

* Referred by Dr. L. L. Grossmann.

Many years ago the patient claimed she had typhoid fever which resulted in partial permanent deafness.

Examination. The patient was a white, well nourished, somewhat dehydrated female appearing subacutely ill. The temperature was 100.6° F., pulse 104, respirations 24 per minute, and blood pressure 148/90. The patient wore a corrective auditory appliance for a moderately severe degree of deafness. Transient râles were noted in the right lower chest posteriorly. There was no evidence of hepatosplenomegaly nor evidence of lymphadenopathy. The clinical impression on admittance to the hospital was a virus infection.

Laboratory Data. The blood hemoglobin on entrance to the hospital was 15.5 grams and the white blood cell count was 13,750 with 81 per cent polymorphonuclears. The blood Kline reaction was negative. Examination of the urine showed 10 to 12 leukocytes and 4 to 6 red blood cells per high power microscopic field. Sedimentation rate determinations showed 73 per cent in fifteen minutes, 44 per cent in thirty minutes and 41 per cent in one hour.

The roentgenogram of the chest (Fig. 6) dated May 20, 1943, showed faint bronchopneumonic infiltrations in the right base. Roentgen examination of the skull and paranasal sinuses was essentially negative.

Course. The course in the hospital was uneventful and the patient's only complaint was marked weakness. There were no complaints referable to the chest. During the first week of hospitalization the temperature ranged between 99° and 102° F. The fever began to fall after the first week in the hospital and when the patient was discharged on May 27, 1943, twelve days after admission, the temperature was normal.

Agglutination tests were negative for typhoid and paratyphoid groups. A blood culture started May 17, 1943, showed no growth for seven days.

Further questioning of the patient yielded the information that the patient's husband was a dealer in domestic pigeons. On May 21, 1943, therefore, blood serum was sent to the U.S. Public Health Service, Washington, D. C., for psittacosis agglutination tests. The results reported were a 4 plus reaction in a dilution of 1:32, 3 plus in a dilution of 1:64 and 2 plus in a dilution of 1:128. Another sample of serum was sent out on June 19, 1943, and the tests showed a 4 plus reaction in a dilution of 1:64, and a 3 plus reaction in a dilution of 1:128. This definite increase in titer over that of the specimen which was sent one month previously justified the laboratory diagnosis of ornithosis.

The patient's husband refused to have blood taken for complement fixation tests for ornithosis.

COMMENT

Meyer states that close to fifty species belonging to five large orders in class Aves may spontaneously be infected with the psittacosis virus. Therefore, we believe that Meyer's[1] suggestion as to the proper terminology of this disease should be adopted. The term psittacosis should denote infections with a proved virus of psittacine origin while ornithosis should include human and bird infections due to psittacosis-like agents of non-psittacine derivation.

Complement fixation tests have proved that many of the influenza-like, mild, atypical infections are due to ornithosis. Meyer states that frank human infections are infrequent because (1) insufficient amounts of the virus are discharged except by ill pigeons and (2) the viruses are of low pathogenicity. The disease is usually transmitted by sick birds but human to human transmission of this disease occurs. Second attacks of ornithosis are also possible.

The incubation period is seven to fourteen days with signs of a generalized infection developing rapidly, thereafter. In the early stages the typhoidal state is simulated, the pulse usually being relatively slow. Clinically, Favour classifies the disease into three types. The disease may occur in a very mild form and last one to two weeks. The pulmonary involvement is usually represented by a central pneumonia. Our third patient (Mrs. M. F.), an example of the mild form of ornithosis, however, showed faint bronchopneumonic infiltrations in the right lower lung.

The moderately severe type of ornithosis is the typhoidal state where the clinical course is more prolonged, usually lasting

three to four weeks. The pulmonary involvement is usually central at first, then a migrating pneumonitis. Our second case (Mrs. R. R.) showed early in the course of the disease a central pneumonic process in the left upper lobe. Subsequently the patient developed more extensive involvement of the left upper lobe, then extension into the right lung and left base.

Mortalities occur in the third and most severe type of ornithosis. This group shows about 30 per cent mortality. The course is more prolonged and widespread pneumonic involvement is characteristic. Mr. B. L., Case I, showed widespread pneumonic involvement with a pleural effusion. In addition, this patient complained of polyarthritis and peripheral lymphadenopathy and later suffered a relapse, the latter being not infrequently observed in this disease.

Clinically speaking, the most striking feature of this disease is the comparative lack of chest complaints in the presence of often widespread pneumonic changes. The subjective complaints are mainly those of weakness and malaise, simulating influenza. Cyanosis is often out of proportion to the amount of lung involvement. Abdominal distention might occur as it did in one of our patients but no localizing signs are present. Polyarthritis and pleural effusions are quite rare.

The white blood cell count is usually low or within normal limits but in 2 of our cases it was slightly to markedly increased. Perhaps secondary infection was responsible for the elevated white blood cell counts in these 2 cases.

If evidence of contact with infected birds becomes established one might suspect an ornithotic infection but confirmation by complement fixation tests for ornithosis is necessary. It is also perhaps advisable to follow the rather rigid criteria laid down by Smadel in order to prove ornithotic etiology. These criteria are (1) isolation of virus during the acute phase of the disease, or (2) the demonstration of the appearance of complement fixing antibodies during convalescence or of a significant rise in titer of these antibodies during the period of recovery. Smadel attaches significance to the results of the complement fixation tests only if there is a fourfold or more rise in antibody titer during convalescence.

Like the clinical aspects of the disease, the roentgen appearance of the chest in ornithosis is similar to that of human atypical pneumonia. The typical case of ornithosis, as Favour describes it, presents a central, patchy, migrating pneumonitis. Case II started as a central pneumonic process in the left upper lobe and later involved the entire left upper lobe, the right lower lung and the left base. Case III, although examined only on one occasion, showed bronchopneumonic areas in the right base. Case I showed bilateral pulmonary involvement with a left pleural effusion.

Roentgenographically it might be difficult and perhaps impossible, early in the course of the disease, to differentiate between ornithotic pneumonia, early lung abscess, early lobar pneumonia or a mediastinal new growth. Serial roentgenograms of the chest, more thorough questioning of the patient, the clinical course of the disease and the all important complement fixation tests for ornithosis will make differentiation possible.

One of our patients (Case II) received a few deep roentgen treatments to the chest. No apparent effect on the clinical course or pneumonic process was noted.

SUMMARY

Three cases of ornithosis with demonstrable pulmonary changes are presented. These examples of ornithosis are presented in an effort to emphasize the fact that many cases of so-called atypical pneumonia, etiology unknown, are actually due to a psittacine or psittacine-like virus; the frequency of such an infection is undoubtedly underestimated. Complement fixation tests for ornithosis should be performed in all

cases of atypical pneumonia even in the absence of known contact with infected or sick birds.

CONCLUSION

The term "primary atypical pneumonia, etiology unknown" should be reserved for cases of atypical pneumonia only after they have been thoroughly investigated from all etiological standpoints including complement fixation tests for ornithosis.

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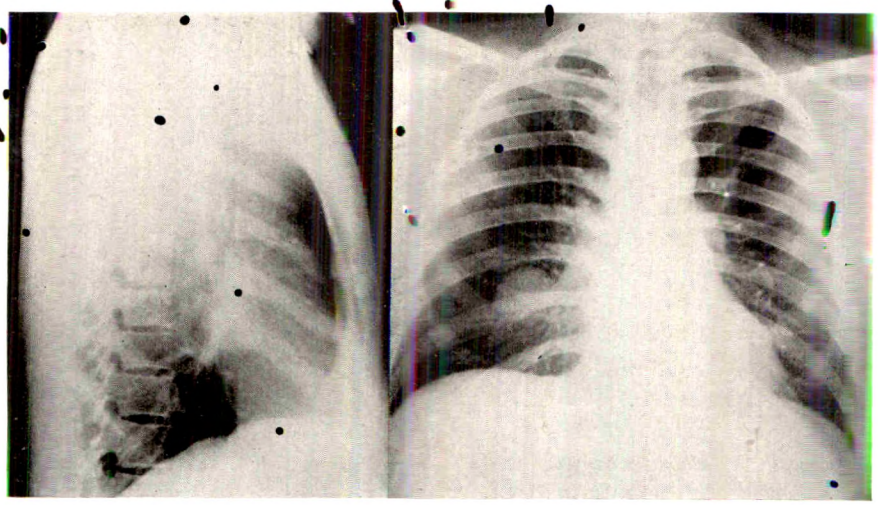


FIG. 3. Carcinoma originating in the secondary bronchi of the right middle lobe.

Figure 1 is a diagram of the normal anatomy of the lungs demonstrating the four lobe concept of the lung anatomy. On the right, the upper and middle and lower lobes are illustrated and the fourth lobe is shown as the apex of the lower lobe. Since the apices of the lower lobes have a separate bronchus and are often the seat of pulmonary carcinoma, their distinction as a fourth lobe is justified. The lingula of the left upper lobe corresponds to the middle lobe on the right.

The accompanying reproductions of roentgenograms illustrate the findings when the bronchi to the various lobes are the site of carcinoma.

Figures 2 and 3 represent 2 cases of the peripheral type. These are involvements of

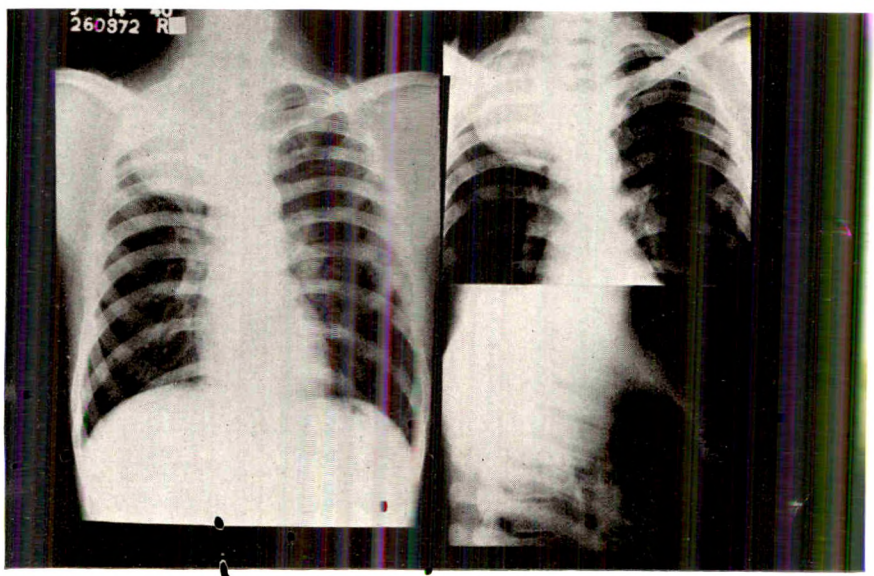


FIG. 2. A superior sulcus type of epidermoid carcinoma in a male, aged thirty-four. This patient complained of shoulder and back pain and presented a Horner's syndrome.

thoracotomies consisted of drainage of lung abscesses or empyema or exploratory operations at which inoperable cancer was encountered.

Although only 7 aspiration biopsies were attempted the record of 5 positive results makes this procedure very valuable, as Craver has already emphasized. It must be realized that the chief limitation of aspiration biopsy is the technical difficulty of obtaining tissue from central lesions which are much more common than those developing in the periphery. But herein lies also the chief value of aspiration biopsy for it can be used to secure tissue where bronchoscopy fails.

Roentgenographically, we have been able to divide these cases into five groups, depending on the location of the carcinoma. By far the most common is the central type which made up 70 per cent of our cases and which originates in the main bronchi and their primary branches. The first signs and symptoms of these tumors are those associated with stenosis of the large bronchi producing collapse pneumonitis and suppuration of the lung distal to the bronchial occlusion. Roentgenographically these produce varying appearances depending on the bronchus involved.

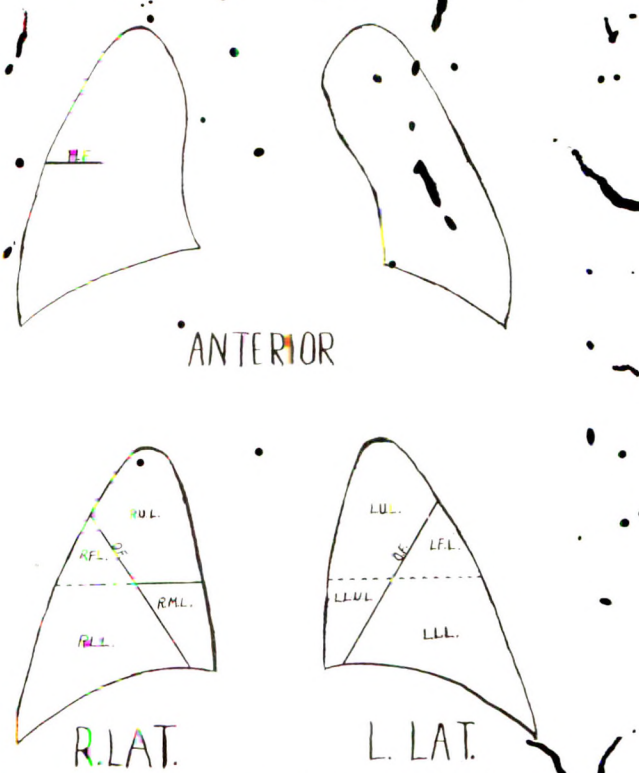


FIG. 1. Diagram of the normal anatomy of the lungs demonstrating the four lobe concept of lung anatomy. *R.U.L.*, right upper lobe; *R.M.L.*, right middle lobe; *R.L.L.*, right lower lobe; *H.F.*, horizontal fissure; *L.U.L.*, left upper lobe; *L.L.U.L.*, lingula of the left upper lobe; *L.L.L.*, left lower lobe; *R.F.L.*, right fourth lobe; *L.F.L.*, left fourth lobe; *O.F.*, oblique fissure.

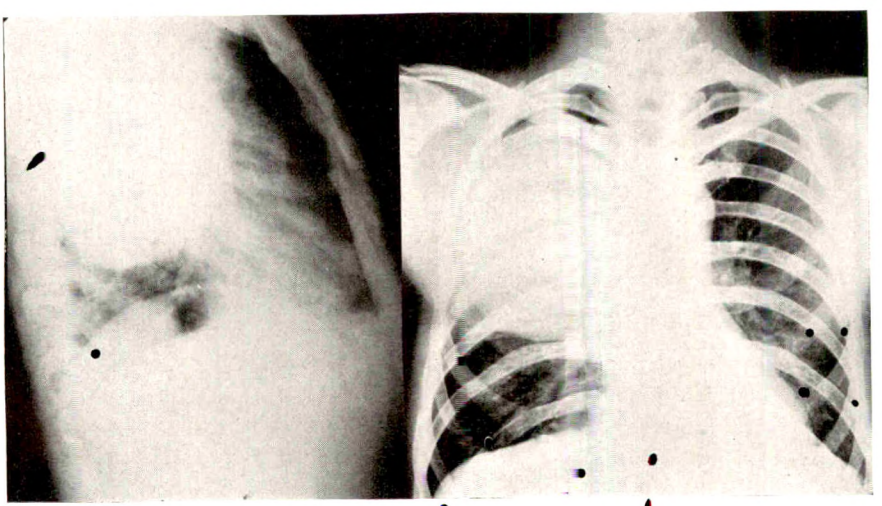


FIG. 2. A circumscribed primary epidermoid carcinoma of the right upper lobe in a white male, aged forty-four.

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PRIMARY CANCER OF THE LUNGS*

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IN VIEW of the increasing incidence of cancer of the lungs and the improved methods of diagnosis now in universal use, the value of keeping this subject before the medical eye seems to be of increasing importance.

This is primarily a statistical study of the 260 cases of cancer of the lung that have been cared for at New York Hospital since 1932 and a detailed account of 100 of these patients with a tissue diagnosis of primary carcinoma of the lung obtained either by biopsy or at postmortem. For analysis, the postmortem diagnosis is given a preference over all other procedures where a tissue diagnosis has been obtained by more than one means.

Of these 100 cases (Table I) 62 were examined at necropsy, 25 were diagnosed by bronchoscopic biopsy and 7 were proved by the examination of tissue removed at thoracotomy. Five cases were diagnosed by microscopic examination of tissue obtained by an aspiration biopsy. Of the 160 cases excluded from this study, the majority were probably primary lung tumors but no histological proof was available.

Table II shows the sex incidence and the age groups of the 100 cases. There were 88 males and 12 females, which is a greater preponderance of the disease in males than reported by Halpert[1] who found a 5 to 1 ratio of males to females affected, but not as high as 90.3 per cent of males that Craver[2] reported in his series of 175 cases.

The analysis of age groups reveals 36 per cent in the sixth decade but the fifth decade shows 32 per cent, which indicates that the disease occurs in relatively young individuals. The average age at the time of admission was 52.5 and the extremes were twenty-seven and seventy-eight years.

TABLE I

DIAGNOSTIC PROCEDURES

Cases analyzed	260
Cases proved by tissue examination	100
Cases examined postmortem	62
Cases diagnosed by bronchoscopic biopsies	25
Cases diagnosed by thoracotomy	7
Cases diagnosed by aspiration biopsy	5

TABLE II

SEX INCIDENCE AND AGE GROUPS

Males	88
Females	12
Age Groups	
20–29	1
30–39	8
40–49	32
50–59	36
60–69	20
70–79	3

TABLE III

SIGNS AND SYMPTOMS

Cough	84
Fever	81
Weight loss	72
Dyspnea	53
Weakness	54
Pain in chest	51
Hemoptysis	43
Anorexia	47
Pulmonary osteoarthropathy	31
Effusion	27
Vomiting	17
Cyanosis	16
Chills	15
Paralysis of diaphragm	14
Nausea	13
Horner's syndrome	7

* Material obtained when the authors were attached to the Department of Radiology, New York Hospital, New York, New York.

Table III shows the signs and symptoms presented by these 100 patients. There has been no attempt to list these chronologically but they were the signs and symptoms presented at the time of the patients' admission to the hospital; cough, fever, and weight loss were the most common complaints which explains why many of the patients with carcinoma of the lung come to a general hospital as a diagnostic problem forcing the clinician to turn to such aids as roentgenology, bronchoscopy and aspiration biopsy to solve the problem.

TABLE IV

DIAGNOSTIC PROCEDURES

Cases bronchoscoped	55
Positive tissues obtained	39
Cases roentgenographed	100
Cases suspected by roentgen ray	86
Cases not suspected by roentgen ray	14
Thoracotomies	32
Curative procedures attempted	7
Aspiration biopsies attempted	7
Aspiration biopsies positive	5
CASES CORRECTLY DIAGNOSED	96

TABLE V

ROENTGENOGRAPHIC TYPES

Main and primary bronchial	70
Peripheral	12
Superior sulcus	8
Segmental	5
Diffuse	2

Three cases did not produce roentgen evidence

TABLE VI

CHEST COMPLICATIONS

Pneumonia	34
Lung abscess	32
Bronchiectasis	15
Pleural effusion	15
Empyema	14
Bronchopleural fistula	8

TABLE VII

COURSE

Average duration of life from first symptom until death	8.1 mo.
Cases examined postmortem	62
Cases known dead without postmortem	31
Cases living	5
Cases lost to follow-up	2

In this entire list of signs and symptoms the only definitely localizing information we have is paralysis of the diaphragms, which is usually a late finding, and Horner's syndrome, which occurred only in superior sulcus tumors. Horner's syndrome was found 7 times in 8 tumors of the Pancoast type and was accompanied by pain in the shoulder and arm of the affected side in 6 of the patients. Three patients who had carcinoma of the upper lobe which were not of the superior sulcus type had shoulder and arm pain and 1 patient with a left lower lobe bronchus tumor had right shoulder pain.

In a few cases the metastases were responsible for the pain at the time of admission. Two of the 3 patients with headache had cerebral metastases, 1 with abdominal pain had liver and pleural metastases and 1 with back pain had vertebral involvement. There was 1 patient with generalized pain produced by widespread metastases.

Table IV lists the diagnostic procedures employed and the results obtained.

In 39 of the 55 cases bronchoscoped, tissue was obtained which revealed carcinoma on microscopic examination. This represents positive results in 71 per cent of the cases done and 39 per cent of the proved cases. But it shows that positive bronchoscopic biopsies were obtained in only 15 per cent of 260 cases available for study at New York Hospital since 1932. Lack of physical signs pointing to bronchial obstruction and masking of the obstruction by complications is probably the reason why so few bronchoscopies were done.

Roentgenography is an extremely important diagnostic procedure. It is, however, unlike bronchoscopy in that it is never absolutely diagnostic but usually suggestive. There were 14 of our cases in which primary carcinoma was not suspected at the roentgenographic examination

Thoracotomy was done in 32 cases. Seven curative procedures were attempted and 2 of these can be considered to have been ultimately successful. The other 25

one or more segments of a lobe caused by carcinoma originating in an intermediate bronchus.

Figure 4 shows a superior sulcus type of carcinoma of the lung which has characteristically invaded the ribs.

Figure 5 is one of the two diffuse tumors of the series which proved to be primary cancer of the lung at postmortem examination.

Figures 6 to 11 inclusive show cancer in the various lobes producing atelectasis of the lung tissue distal to the involved bronchus.

The 3 cases which did not produce roentgen evidence of a pathological condition were very small interbronchial tumors discovered at postmortem in patients dying of another disease.

The course of the disease in this series is anything but encouraging for it has already claimed the lives of 93 per cent of its victims and there are 2 cases which have been lost to follow-up but are probably dead. Five of the patients are still living but 3 of these are relatively recent cases of which 2 have had thoracotomies which proved inoperable, and the third is in a home for incurables.

The average duration of life, for the patients who have died, was 245 days from the onset of the disease.

There are 2 cases who have had pneumonectomies and are alive and well three and four years after operation so they can be considered free of disease.

Table VIII presents the distribution of metastases in the 62 autopsied cases.

The regional lymph nodes were involved most often by metastases but the other common sites were the pleura, bone and brain.

There were 7 cases which had no metastases at postmortem; these included 3 small interbronchial lesions which were incidental autopsy findings in patients dying of other causes. The remaining 4 cases without metastases were patients with peripheral carcinomas. It is noticeable that Babin and Neuhof[3] found the parenchymal carcinomas more apt to be circumscribed and slower to metastasize, for the only cases which had reached an appreciable size and had not spread elsewhere were of the peripheral type.

In this series the only treatment of carcinoma of the lung that could be considered curative was pneumonectomy, but recent-

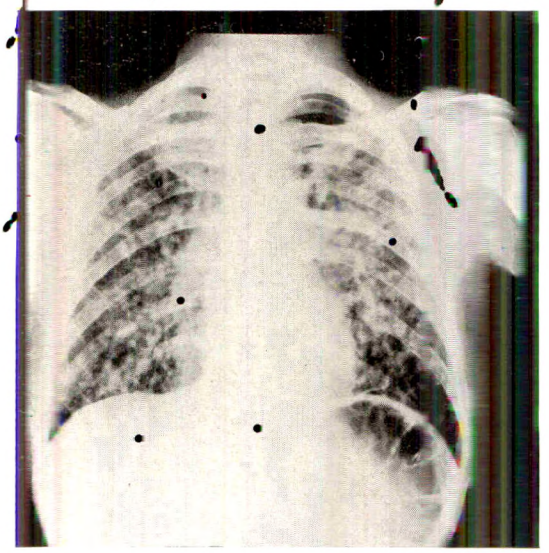


Fig. 5. Diffuse bilateral involvement of the lungs with carcinoma which at autopsy was proved to be primary in the lungs.

Table VIII

Metastases

Of the 62 autopsied cases	40 complete 16 all except head 6 limited to chest	
No metastases		7
Bronchial nodes		30
Mediastinal nodes		29
Pleural metastasis		22
Bone		20
Adrenal		18
Retroperitoneal nodes		16
Liver		14
Brain (40 cases)		11
Kidney		10
Cervical nodes		7
Heart		5
Esophagus		4
Spleen		4
Lung		4

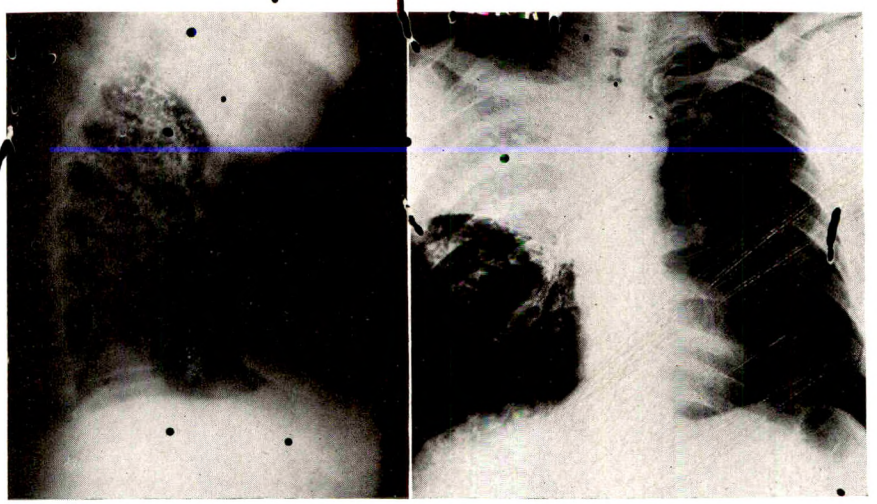


FIG. 6. Atelectasis of the right upper lobe secondary to a carcinoma obstructing the bronchus.

gen therapy was resorted to in 34 cases with apparent symptomatic benefit in 10 of these. The blocked bronchus was opened allowing aeration and drainage of the atelectatic and secondarily infected lobe. The average duration of life after the first symptoms in the cases treated with roentgen irradiation was 310 days, or sixty-five days longer than for those for the entire group (see Table IX).

TABLE IX

ROENTGEN THERAPY

Cases treated	34
Expected course not altered	24
Benefited	10
Average duration of life in cases receiving radiation therapy	$10\frac{1}{4}$ mo.

The pathological classification is that submitted by Dr. Dan McEndy of the Department of Pathology at New York Hospital who reviewed and classified all the tissue examined at autopsy. His classification is similar to that used by Jenkinson

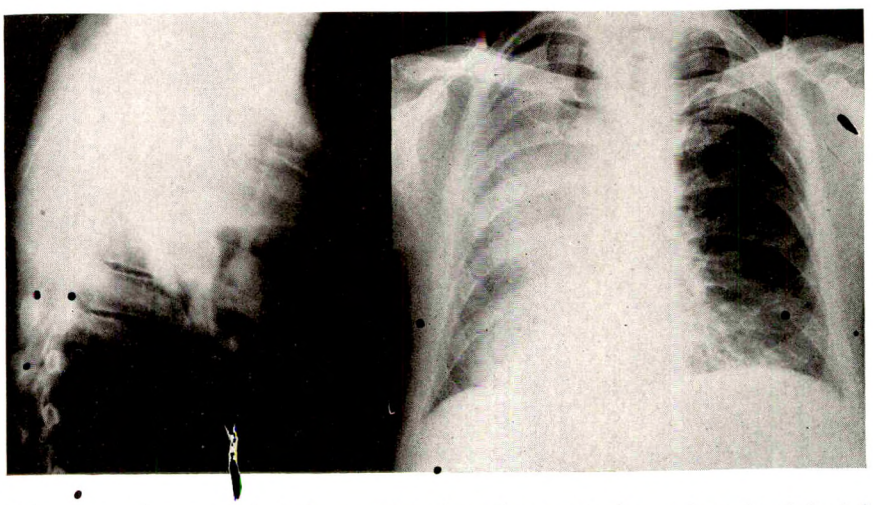


FIG. 7. Primary carcinoma of the left upper lobe bronchus producing atelectasis of the left upper lobe. The tumor mass can be seen at the apex of the atelectatic portion of the lung.

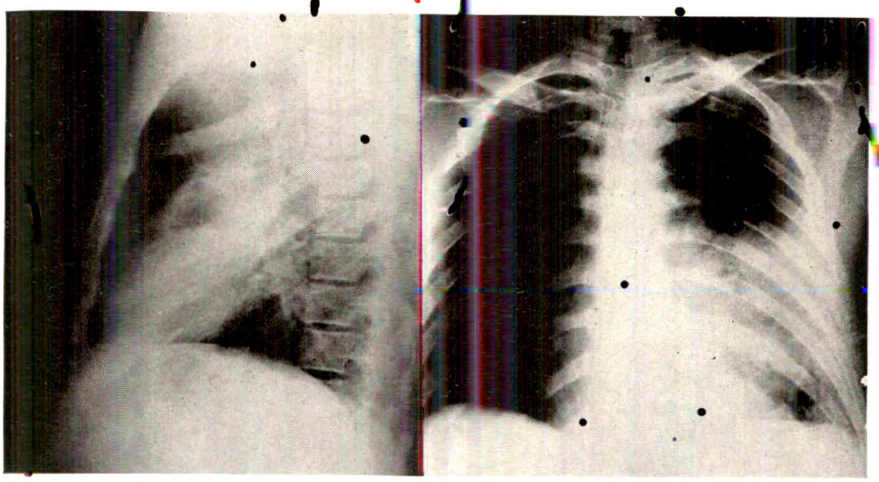


FIG. 8. Carcinoma obstructing the bronchus of the lingula of the left upper lobe. Note the atelectasis of the lingula of the left upper lobe.

and Hunter.[4] The tumors are divided into three major groups, as follows:

1. Epidermoid or squamous carcinoma which incluces only those cases in which obvious keratin and/or epithelial pearls are present.

2. Adenocarcinoma—those which form acini and/or produce mucin.

3. Undifferentiated—which includes the typical forms such as "small cell carcinoma" of the oat cell type as well as those which show some differentiation but not enough to place them in a specific category of epidermoid or adenocarcinoma.

TABLE X

MICROSCOPIC PATHOLOGICAL CLASSIFICATION

Epidermoid carcinoma	50%
Adenocarcinoma	18%
Undifferentiated	32%

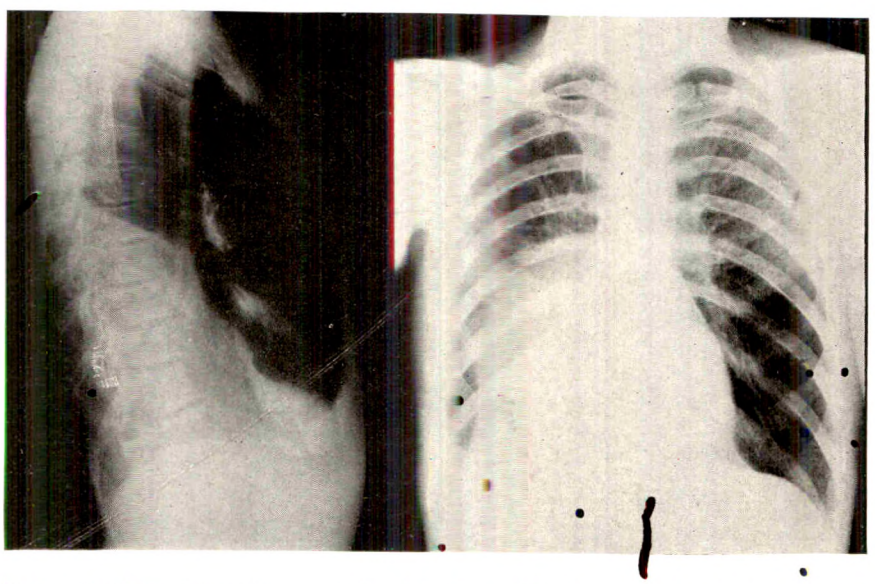


FIG. 9. Epidermoid carcinoma of the right lower lobe bronchus producing a typical atelectasis of the lobe it supplies.

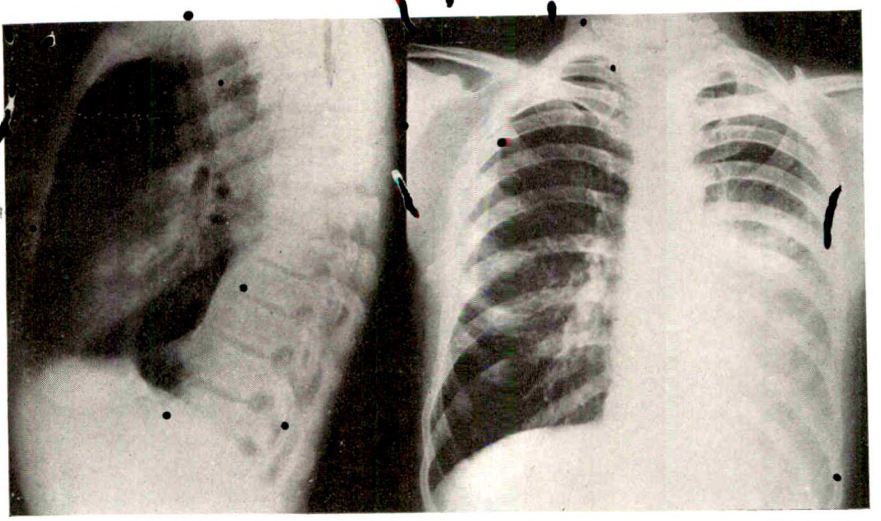


FIG. 10. Carcinoma of left lower lobe bronchus producing atelectasis of the entire left lower lobe with shifting of the mediastinum toward the affected side.

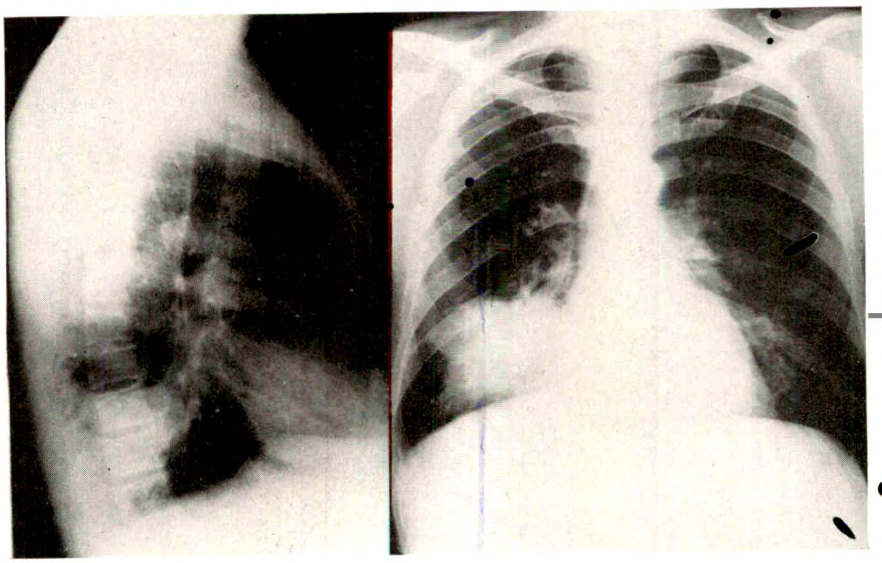


FIG. 11. A peripheral carcinoma of the right lower lobe in a white male, aged sixty-four. The patient is well four years after a pneumonectomy.

As shown in Table X, in this series 50 per cent were of the epidermoid type, 18 per cent were adenocarcinomas including 2 mucous gland carcinomas. The remaining 32 per cent were placed in the undifferentiated group.

CONCLUSIONS

1. Bronchogenic cancer is a major problem far more serious than is generally appreciated.

2. Every effort should be made to establish the exact diagnosis by procurement of tissue for histological examination.

3. The problem, as in all malignant disease, is early diagnosis. Fifty per cent of the cases we see should be subjected to surgery and this surgery should be extremely radi

cal with no effort made to hold down the surgical mortality at the expense of radical curative surgical procedures.

4. The clinician who sees patients with symptoms referable to the chest should use the roentgenologist, bronchoscopist and surgeon early and urge radical surgery on the patient.

We are indebted to Dr. Dan Tucker who assisted us with the review of these cases and presented the outline of our work to the Research Society of New York Hospital; to Dr. N. C. Foote and Dr. Dudley Blossom* of the Department of Surgical Pathology of New York Hospital for reviewing the microscopic pathology of this series.

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THE ROENTGEN VISUALIZATION OF TUMORS OF THE CARDIA.

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PROMINENT among the gastric lesions which elude early diagnosis is cancer of the cardiac end of the stomach. Its presence is frequently unsuspected both by the clinician and by the roentgenologist, and unless carefully sought for in each gastric study may pass undiagnosed only to be identified some time later when dysphagia, cardiospasm, and other significant signs and symptoms appear. Many of us have had the embarrassing experience of demonstrating a tumor of the cardia on roentgenograms previously reported as negative. The demonstration of a large filling defect in the upper portion of the stomach, esophageal obstruction, or rigidity of the lesser curvature superiorly readily serves to establish the diagnosis, but we have always felt it desirable to develop a method for earlier and more definite identification of these growths.

Stewart and Illick[1] advise close observation of the first swallow of barium as the stream enters the stomach. They describe several findings indicative of cancer at the cardia including dilatation or other evidence of esophageal obstruction, distortion of the rugal pattern, deformity of the gas bubble and occasionally the shadow of a soft tissue mass in it. Kirklin[2] justly stresses the identification of a tumor mass in the gas bubble of the empty or partially filled stomach, and cautions against relying on roentgenograms of the completely filled stomach or roentgenograms made with the patient in the Trendelenburg position. The use of Seidlitz powders better to visualize the cardia has its advocates.

Before the ingestion of a liquid barium mixture, the normal gas bubble is half moon in shape and varies considerably from time to time in the same individual. The base, formed by fluid in the stomach, may measure from 4 to 10 cm. in length, and its average height varies from 3 to 8 cm. The upper rim lies adjacent to the left leaf of the diaphragm, and the inner inferior angle is in close proximity to the medial left diaphragmatic crus. The fluid level is usually at or slightly above the cardio-esophageal orifice. In some patients the gas-filled splenic flexure lies lateral and occasionally anterolateral to the bubble. Infrequently the splenic flexure is interposed between the diaphragm and the stomach bubble. Occasionally the latter is unusually large, as may occur with aerophagia. We have on occasion noted a considerable amount of gas in the stomach on six hour roentgenograms taken with the patient in the prone position, sometimes sufficient to outline a tumor mass in the cardia. The stomach beneath the gas bubble cannot be identified on direct studies of the abdomen, as a rule, because the gastric walls and contents present a roentgenographic density indistinguishable from the surrounding soft tissues.

Obviously the most reliable index for the correct diagnosis of tumors of the cardia is a clear demonstration of the tumor mass itself. Sometimes the existing stomach bubble is sufficient for this purpose. At other times, when there is insufficient gas to be of value, an adequate bubble can be created by air insufflation. This technique has already been applied to roentgen examination of the colon, bladder, and brain, so that the air contrast enemas, cysto-aerograms and pneumo-encephalograms have a well established position in diagnostic roentgenology.

In selected cases we have been able to produce contrast roentgenograms of the cardia by inflating the stomach through a Levin tube under roentgenoscopic control, using the hand bulb from a blood pressure apparatus. The amount of air necessary varies considerably, depending on the size of the cardia and the degree of distention desired. The insufflation may be made with the stomach empty or partially filled with

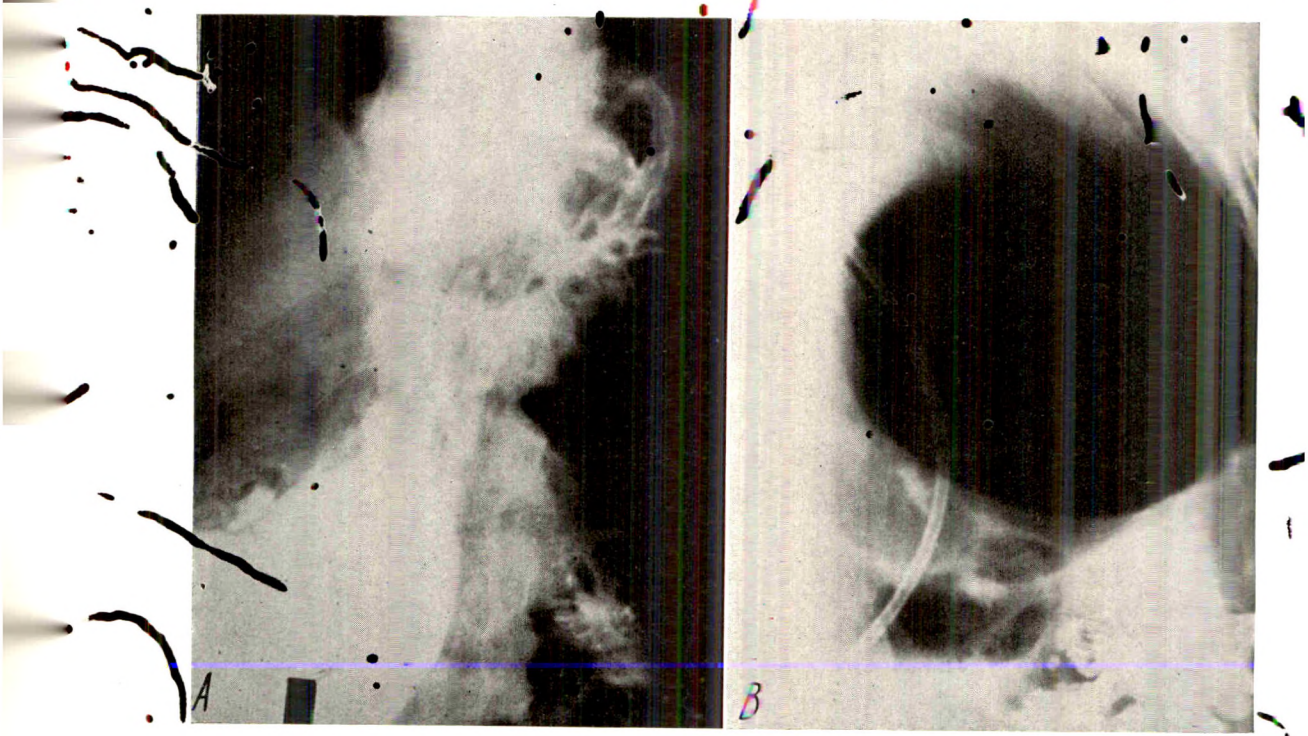


FIG. 1. Case 1. *A*, defect from spinal pressure in the mesal aspect of the cardia, with apparent luminal encroachment simulating tumor. *B*, same as *A* after insufflation of the cardia, revealing the latter to be normal.

barium. In suspicious cases the patient first is given a teaspoonful of thick barium paste, followed by 6 ounces of a liquid barium mixture. After roentgenoscopic study, roentgenograms are made with the patient in the erect and supine positions in the posteroanterior, oblique and lateral projections as indicated by the preceding roentgenoscopy. Roentgenograms made with a technique slightly less penetrating than the usual gastric exposure may demonstrate tumors otherwise overlooked.

The examination, which is adaptable to both office and hospital practice, can be performed rapidly and with a minimum of discomfort to the patient. We have encountered no untoward reactions from the air insufflations. This method is of special value for detecting masses beneath and extending slightly above the fluid level of the stomach bubble and in examining patients with cascade stomachs.

Precautions should be taken to avoid interpreting extraneous shadows as intrinsic gastric masses. Among these are the shadows of an overlying spine, of an enlarged left ventricle, a high retroperitoneal tumor, an exudate in the left lumbar region, and, in rare instances, an aneurysm of the upper abdominal aorta. An enlarged left lobe of the liver may offer some difficulty, but can usually be identified by the smoothness of its borders, its lingual contour and its continuity with the main hepatic mass. A tumor is usually portrayed as an irregular lobulated mass or mass independent of the hepatic shadow.

Case reports are appended which have been selected to illustrate various diagnostic and technical points.

REPORT OF CASES

CASE 1.—S. S., male, aged sixty-seven, had epigastric cramps for fourteen months before admission, which were relieved somewhat with milk. Roentgenograms taken at the onset of his present illness were reported as negative. During the next six months he had successive faint-

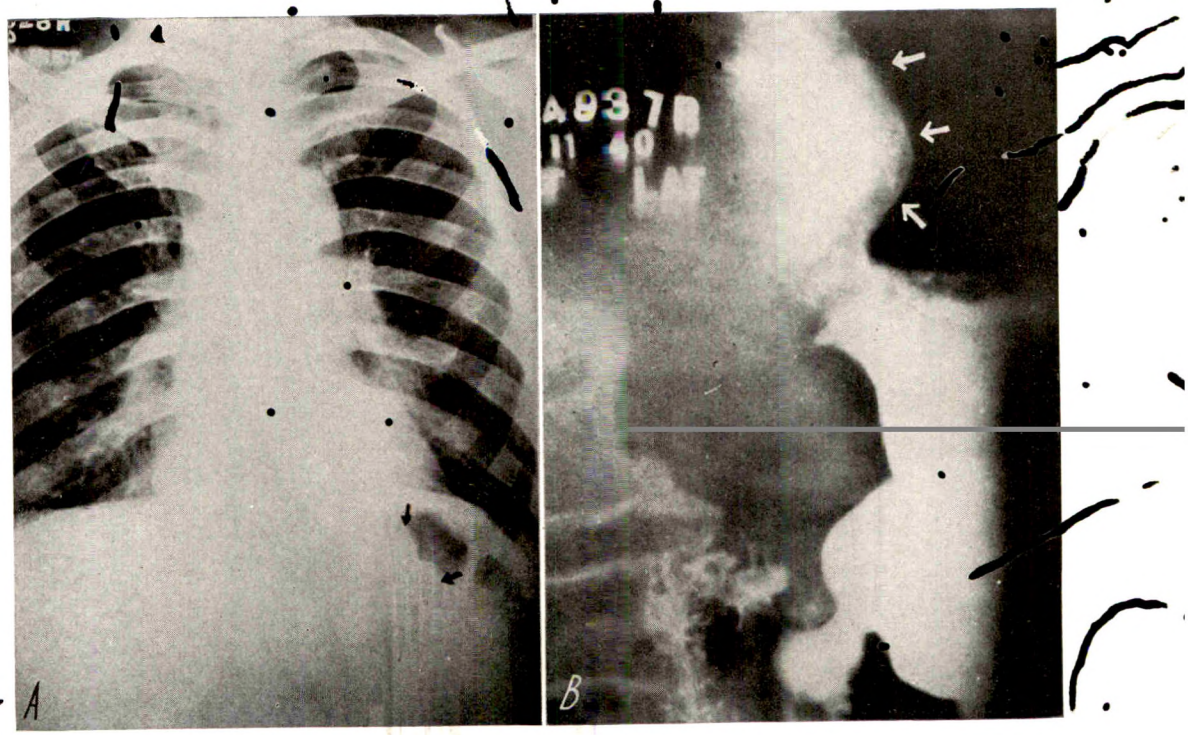


FIG. 2. Case II. *A*, nodular soft tissue shadow encroaching on the gastric lumen at the cardia suggesting hepatic border or tumor. *B*, tumor mass, proved carcinoma, suspected on the chest roentgenogram, *A*, and confirmed by gastric insufflation, lateral projection.

ing spells and a secondary anemia developed. There was no history of melena or hematemesis until one month before admission when he had a tarry stool. His hemoglobin fell from 95 to 55 per cent.

Examination revealed a pronounced pallor. There was the appearance of recent weight loss. His liver, smooth and not tender, was palpated three fingersbreadth beneath the costal margin. There were no palpable abdominal masses. The clinical diagnosis was cancer of the stomach, duodenal ulcer to be ruled out.

Routine roentgenograms revealed a persistent duodenal defect indicative of an ulcer. On the roentgenograms there was incomplete filling of the mesial portion of the cardia suggestive of tumefaction. After air inflation this observation was not confirmed, and the cardia was reported as normal (Fig. 1*A* and *B*).

The patient improved under symptomatic treatment.

Comment. The defect in the cardia on the routine roentgenograms was caused by spinal pressure. The upper half of the stomach was displaced toward the midline and was somewhat narrowed by a redundant splenic flexure. After air insufflation it was apparent that the cardia was normal. In view of the symptomatology and the patient's age, it was essential to rule out the cardia as the site of a malignant tumor.

CASE II. A. K., male, aged sixty-six, was treated intermittently for a peptic ulcer for the past nine years. He felt well under dietary treatment until three months ago when he developed anorexia, weakness and loss in weight of 20 pounds. Before admission, for a two weeks' period, he had had epigastric pain both before and after eating. There was no dysphagia. No positive findings were noted on physical examination. The clinical diagnosis was cancer of the stomach.

A teleroentgenogram of the chest revealed a mass, indistinguishable from the hepatic border, protruding into the stomach gas bubble. Roentgenographic study of the stomach showed free passage of barium through the cardio-esophageal orifice. There appeared to be a lat

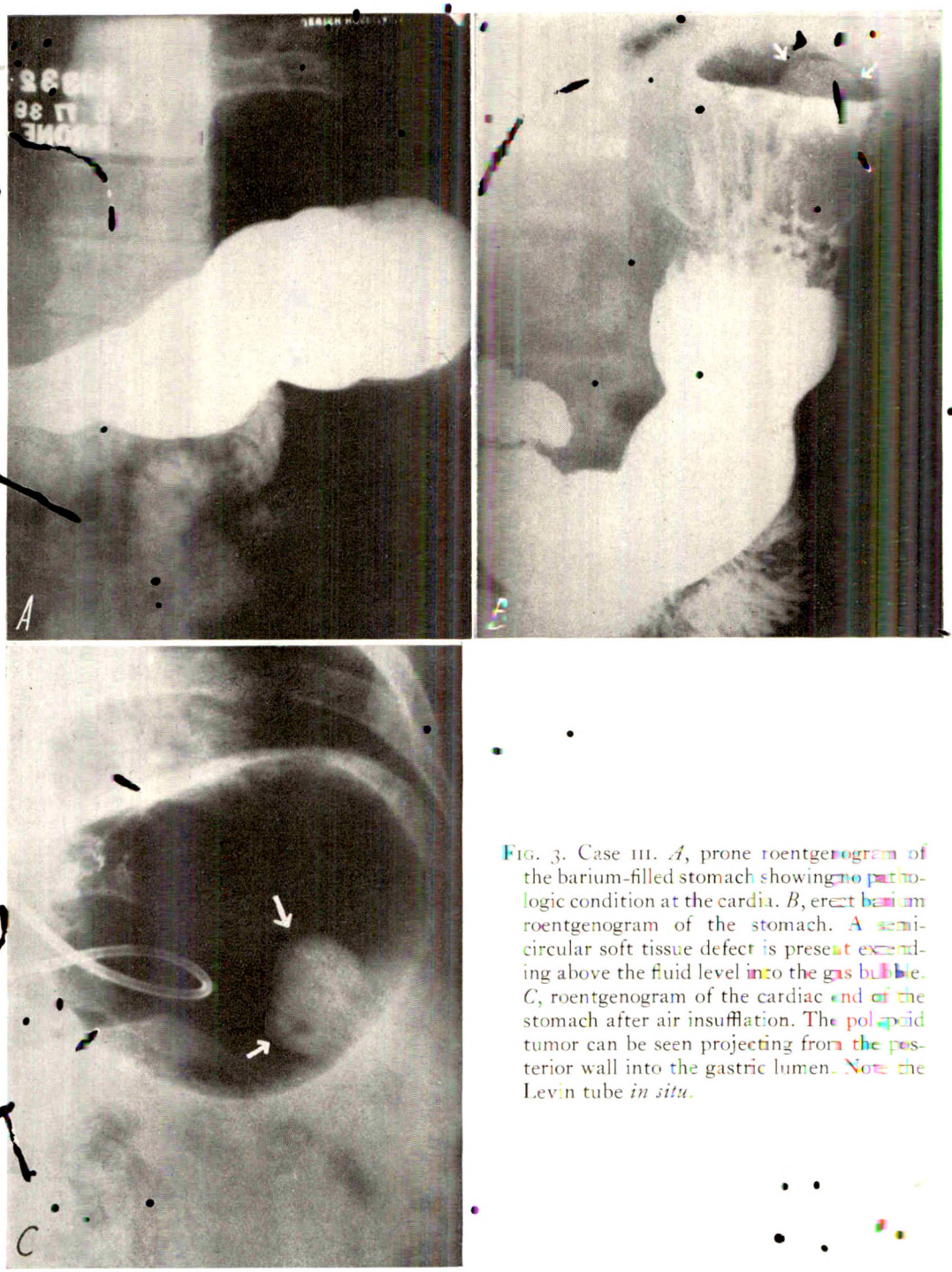


FIG. 3. Case III. *A*, prone roentgenogram of the barium-filled stomach showing no pathologic condition at the cardia. *B*, erect barium roentgenogram of the stomach. A semicircular soft tissue defect is present extending above the fluid level into the gas bubble. *C*, roentgenogram of the cardiac end of the stomach after air insufflation. The polypoid tumor can be seen projecting from the posterior wall into the gastric lumen. Note the Levin tube *in situ*.

real displacement of the barium stream as it entered the stomach. Roentgenograms, by direct study of the barium filled stomach, were normal. After inflation with air, a mass was portrayed as a positive shadow against the radiotransparent air. It was best seen on the roentgenograms taken with less penetration than is usually employed (Fig. 2, *A* and *B*),

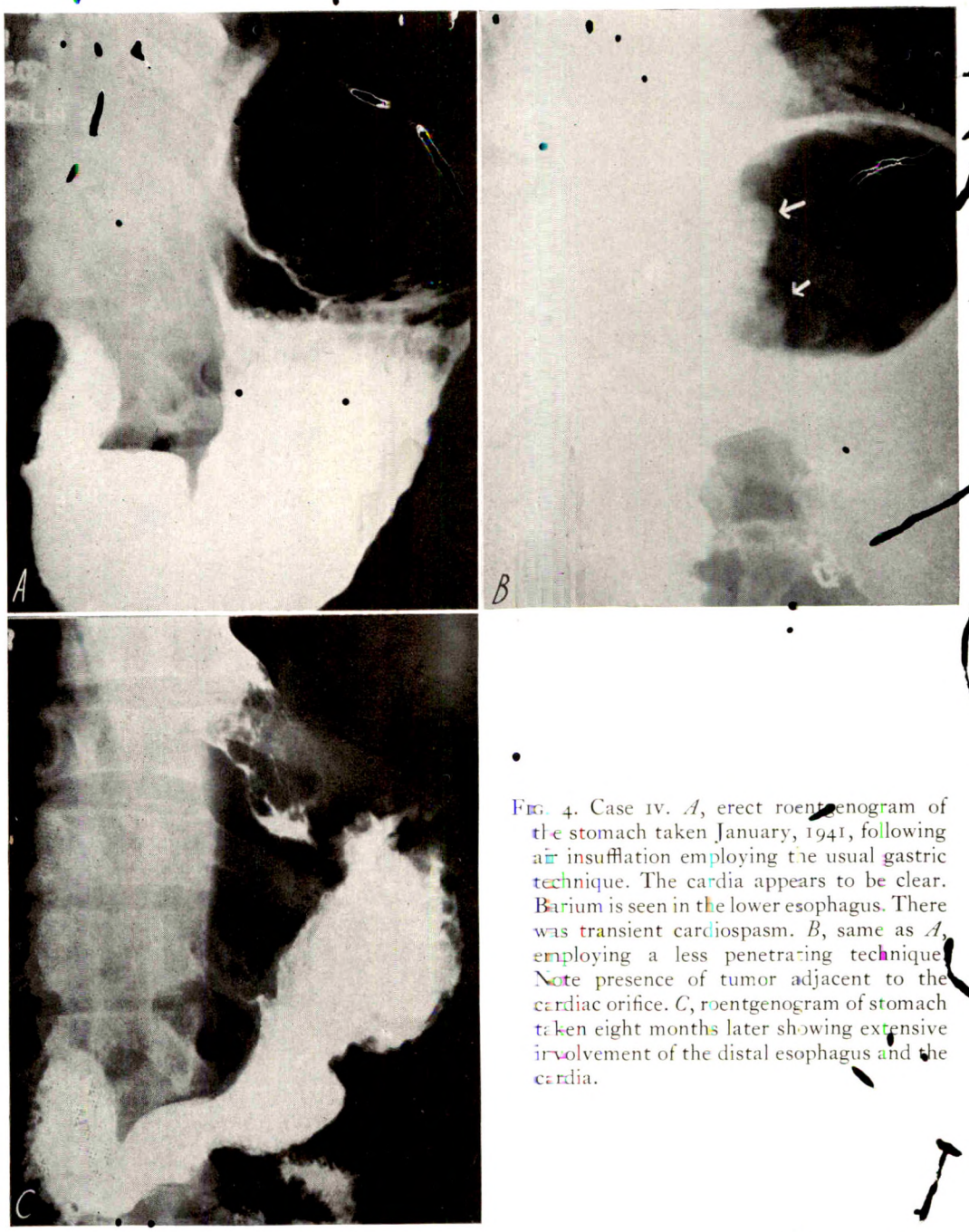


FIG. 4. Case IV. *A*, erect roentgenogram of the stomach taken January, 1941, following air insufflation employing the usual gastric technique. The cardia appears to be clear. Barium is seen in the lower esophagus. There was transient cardiospasm. *B*, same as *A*, employing a less penetrating technique. Note presence of tumor adjacent to the cardiac orifice. *C*, roentgenogram of stomach taken eight months later showing extensive involvement of the distal esophagus and the cardia.

Gastroscopy showed a nodular smooth mass at the cardia. At operation a large inoperable cancer was found encircling the cardia and extending into the diaphragm. Liver metastases were present.

Comment. The diagnosis was suspected on the chest roentgenogram because the stomach bubble happened to be present and the exposure made with sufficient penetration to visualize a soft tissue irregularity. The diagnosis was confirmed by subsequent roentgenograms using the technique of air insufflation to delineate the tumor mass more clearly.

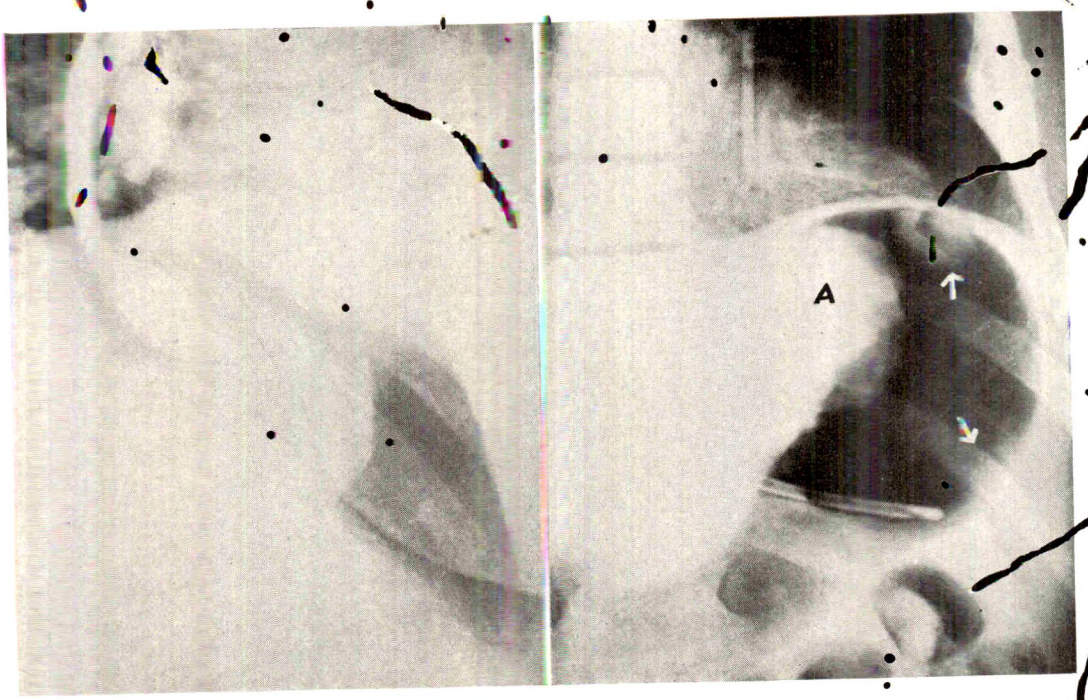


FIG. 5. Case v. Right oblique view of the cardia after air insufflation revealing a large soft tissue mass adjacent to but not involving the cardiac orifice. No evidence of cardiospasm. The esophagus is outlined with barium paste.

FIG. 6. Case VI. Gastric insufflation revealing a large tumor mass (carcinoma) of the cardia, with a secondary deposit on the dome of the stomach, and one on the greater curvature just beyond the tip of the Levin tube.

Gastroscopy showed a firm irregular mass at the cardia. At operation a firm, irregular, slightly nodular tumor, about the size of a lemon, was found apparently arising from the right posterolateral wall of the stomach at the cardio-esophageal opening. The remainder of the stomach was slightly smaller than usual, but was otherwise normal. The liver contained a metastatic nodule, 2 cm. in diameter, on the anterosuperior aspect of the right lobe.

Comment. In this instance the neoplasm of the cardia did not involve the cardio-esophageal opening. Although the patient complained of dysphagia, there were no roentgenologic evidences of esophageal obstruction. The diagnosis was suspected from the roentgenoscopic findings and was confirmed by the air contrast studies.

CASE VI. E. B., male, aged sixty-two, complained of weakness for four years before admission. A diagnosis of secondary anemia was made during this period, and he responded well to a series of liver and iron injections. He was able to continue his work as a painter until one year ago. At that time there was a recurrence of weakness. He developed a marked pallor, lost 30 pounds in weight in the five months prior to admission, and one month ago spit a teaspoonful of bright red blood. There were no gastrointestinal symptoms, and his appetite remained excellent. Physical examination revealed a Virchow's node in the left supraclavicular area. There was no other adenopathy. The spleen was not palpable. The liver was moderately enlarged. Gastric extraction revealed fresh blood on several occasions, and an achlorhydria. Hemoglobin was 40 per cent.

Roentgenograms of the stomach revealed a large filling defect in the cardia. Gastric insufflation portrayed the tumor with better detail, and demonstrated a daughter nodule at the dome of the cardia and another on the greater curvature opposite and slightly below the primary growth. Roentgenographic examination of the chest showed a metastatic deposit in the axillary portion of the left eighth rib (Fig. 6).

CASE III. M. P., male, aged sixty-six, complained of epigastric distress for five years before his admission to the hospital in 1939. Eight weeks before, he had had hematemesis for a twenty-four hour period, followed by tarry stools. This was followed by a pronounced initial weight loss which, however, was soon regained. There was no dysphagia or other subjective complaint. Objectively the examination was negative.

Roentgenograms of the stomach, taken with the patient in the erect position, revealed a semilunate, soft tissue shadow suggestive of a mass protruding into the cardia of the stomach from its posterior wall. Roentgenograms taken in the prone and Trendelenburg positions were negative. Following air insufflation a polypoid tumor mass, about 3 cm. in diameter, protruding into the cardia from a broad base was demonstrated (Fig. 3, *A*, *B* and *C*).

Gastroscopic examination revealed a sessile polyp attached to the posterior wall near the greater curvature. The adjacent walls were pliable, with no evidence of infiltration. The overlying mucosa was irregular, glistening and intact.

On July 17, 1940, the patient was again gastroscoped with similar findings. His condition remained excellent until November, 1940, when another painless hemorrhage occurred. He was again gastroscoped early in 1941 with no change in the findings. On April, 1942, an ulcerated area, covered with a grayish exudate, about 3 cm. in diameter, was seen over the center of the polyp. This apparently healed spontaneously since a subsequent gastroscopy in July, 1942, revealed intact mucosa.

Comment. In this instance the polyp could not be diagnosed on the roentgenograms taken in the prone position, and its presence could only be suspected on the erect studies. The air contrast roentgenograms of the stomach portrayed the lesion with such clarity that there was no doubt concerning the presence of a mass and the nature of its attachment.

CASE IV. M. W., male, aged sixty-one, complained of difficulty in swallowing, weight loss and weakness for three months beginning in January, 1941. Esophagoscopy revealed an irregular beefy-red indurated lesion at the cardiac end of the esophagus. A biopsy specimen was reported as leukoplakia. He was readmitted eight months later with epigastric pain associated with swallowing, increased dysphagia, and vomiting after meals. There had been progressive loss of weight. No significant findings were noted on physical examination.

Roentgenologic examination of the esophagus and stomach at his first admission in January, 1941, revealed some difficulty in the passage of barium at the cardio-esophageal junction. Transient dilatation of the esophagus was present. In view of the suspicion of a neoplasm at the cardia, an air insufflation was performed. On the roentgenograms taken with normal roentgenographic density, the cardia appeared to be normal. However, on roentgenograms made with less penetration it was possible to demonstrate an infiltrating lesion along the lesser curvature extending beyond the cardio-esophageal junction into the cardia. Re-examination in September, 1941, revealed an extensive tumor involving the distal third of the esophagus and a large portion of the cardia (Fig. 4, *A*, *B* and *C*).

At operation a large, firm, fungating mass was found involving the upper portion of the lesser curvature, cardiac orifice, and esophagus. Metastatic lymph nodes were present in the gastrohepatic omentum. A gastrostomy was performed.

Comment. When first seen the lesion was not too dense nor prominent, and might easily have been overlooked on the routine insufflation roentgenograms as it was on the routine roentgenograms if soft tissue technique had not been used.

CASE V. B. R., female, aged fifty-six, swallowed a fragment of bone six months before admission. Fever followed soon thereafter, with pain in the upper chest and neck. An abscess developed which ruptured into the upper esophagus; relief followed. Two weeks later she complained of nausea, epigastric pain and dysphagia. During the following four months there was a loss in weight of 20 pounds. When admitted she could swallow only liquids.

Roentgenologic examination of the esophagus revealed free passage of barium through the cardio-esophageal junction. No evidence of dilatation was present. However, a soft tissue mass was seen protruding into the gas bubble, more clearly demonstrated by air insufflation (Fig. 5).

PARTIAL PARALYSIS OF A HEMIDIAPHRAGM*

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THE motor innervation of the diaphragm is essentially provided by the phrenic nerve. Many authors (Russell, Schlaepfer[8,9]) believe that the phrenic is the only motor nerve of the diaphragm, others (Cavalié,[1] Felix[2]) assume that the intercostal nerves furnish motor branches, too. Also the sympathetic nerves and the vagus may, according to some investigators (Kuré and Shimbo,[4] Felix,[2] Cavalié) participate in the motor innervation of the diaphragm.

Damage to a phrenic nerve resulting in paralysis of a hemidiaphragm is not infrequent. It occurs in injuries and diseases of the cervical cord; in injuries to and operations of the phrenic nerve; in tumors in the region of the neck and the mediastinum and in neuritis. Paralysis of the diaphragm can also be caused by disease of its muscle.

Partial paralysis of a hemidiaphragm, however, is apparently very rare. Hitzenberger[3] reports such a case:

A female patient with cerebrospinal lues had among other symptoms a paresis of her right arm. On roentgenoscopy in the postero-anterior view, the medial portion of the right diaphragm was elevated. In the lateral view its anterior portion appeared elevated. The right diaphragm moved poorly and showed lagging. On sniffing, the medial part moved paradoxically. The lateral and posterior portions of the right diaphragm and the left diaphragm were normal. Hitzenberger believes that that part of the phrenic nerve was damaged which supplies the medial and anterior part of the diaphragm and that such an injury is possible only in the nuclear part of the nerve. Russell[7] found in the macacus that on stimulation of the fourth cervical nerve root the medial part of the diaphragm contracted, the outermost portion of the diaphragm contracted when the sixth root was excited; the intermediate portion when the fifth root was stimulated.

On routine roentgenologic examination a partial paralysis of a diaphragm was discovered accidentally.

A man, aged twenty-nine, had had an anterior poliomyelitis at the age of eleven. All extremities had been involved. He had recovered nearly completely and was able to do any kind of work and sport without difficulty.

On physical examination there was a marked scoliosis of the thoracic spine to the left with marked kyphosis and compensatory lordosis of the lower thoracic and lumbar spine. The neurologic examination (Dr. Leo Spiegel) revealed the following abnormal findings: The volume of the left upper extremity is somewhat diminished compared with the right. The volume of the right lower extremity is somewhat diminished compared with the left. The muscle power in the left upper extremity is diminished. Deep reflexes are practically absent except for an occasional right ankle jerk and a questionable radial reflex bilaterally. The sternocleidomastoid muscle on the left is thinner than the one on the right. Summary: The patient presents a picture compatible with an old anterior poliomyelitis. The brunt of the lesion appears to have been in the cervical cord, predominantly on the left side. There is also a milder involvement of the lower lumbar cord on the right side. An interesting feature is the practically complete areflexia despite the presence of apparently good nutrition in many of the muscles.

Roentgen examination (Fig. 1 and 2) and roentgenoscopy showed the following: There is marked scoliosis in the mid portion of the thoracic spine, with the convexity towards the left. The left diaphragm is normal and shows good excursion. The central two-thirds of the right diaphragm is markedly elevated and moves only slightly with respiration and paradoxically on sniffing. The lateral and posterior part of the right diaphragm moves more freely. The heart is normal in size and shape.

Spirometric studies revealed considerable functional damage. The vital capacity was

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Comment. Although there was little doubt of the correct diagnosis from the routine roentgenograms, those made after air insufflation outlined the tumor more clearly and revealed in addition the presence of two secondary growths.

SUMMARY

The positive roentgenologic diagnosis of cancer of the cardia of the stomach may depend on visualization of the tumor mass itself. At times this may be accomplished by demonstrating the tumor through the existing gas in the stomach. In other instances the diagnosis may be made by creating a large gas bubble by insufflating air into the stomach through a Levin tube. The technique is described and illustrative cases are reported.

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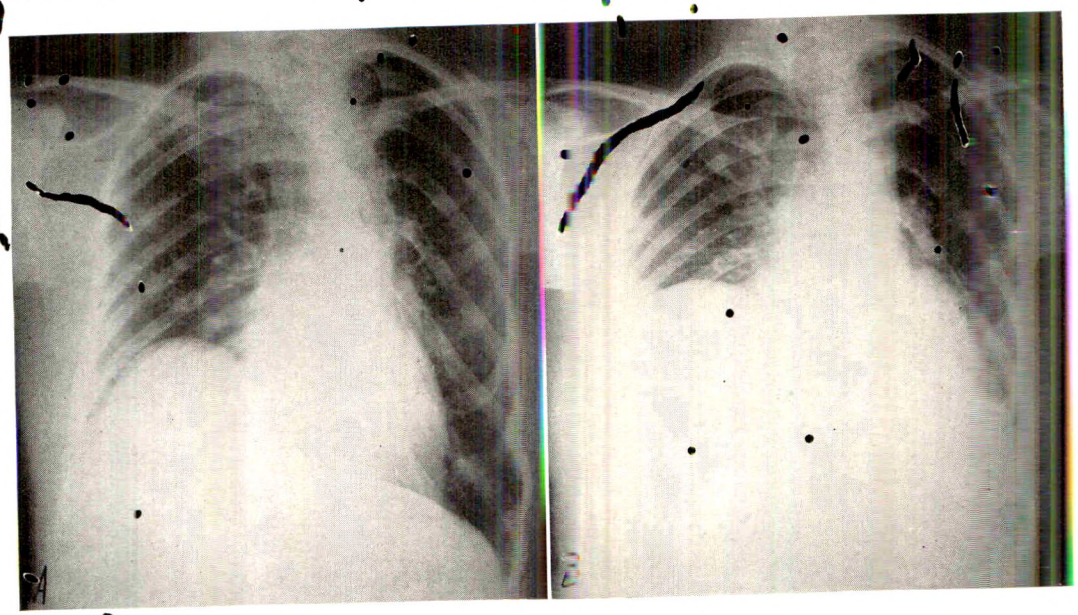


FIG. 1. Posteroanterior view. *A*, inspiration; *B*, expiration. The left diaphragm shows good excursion. The central part of the right diaphragm is elevated and moves only slightly; the lateral part moves more freely.

2,400 cc., that is 60 per cent of normal. The maximum breathing capacity was 53 liters (normally 120 liters or more). The maximum breathing capacity divided through the minute volume of respiration was 8.9 (normally 10 to 15). The oxygen debt after exercise was normal. How much of the reduction in pulmonary volume and function can be attributed to the paralysis of the diaphragm and how much of it is caused by the kyphoscoliosis cannot be decided. The scoliosis of the spine to the other side is a frequent occurrence in paralysis of a diaphragm. In this case it may have been caused also by weakening of other muscles.

How can it be explained that the right diaphragm is partly paralyzed whereas the neurological examination reveals that the left side of the cervical cord seems to be affected more than the right side? Crossing of the phrenic nerves might be the reason. Rosenblueth and Ortiz[6] and Rosenblueth, Klopp and Simeone[5] were able to prove in animals of several species the presence of a crossing of the phrenic in the spinal cord. The partial paralysis, as mentioned above in Hitzenberger's case, is probably due to damage to the nuclear region of that portion of the phrenic which supplies the anterior and medial region.

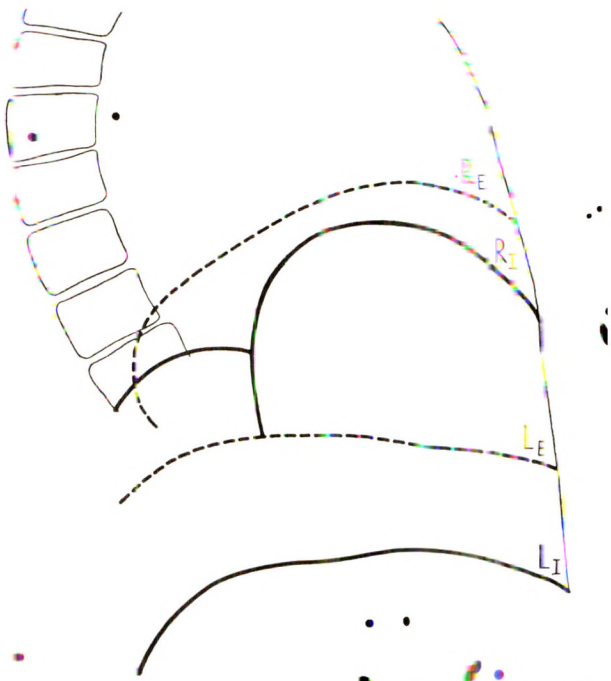


FIG. 2. Right lateral view. Diagram traced from roentgenogram. R_I, R_E, right diaphragm in inspiratory and expiratory position; L_I, L_E, left diaphragm in inspiratory and expiratory position. The left diaphragm shows good excursion. The right diaphragm is elevated; its anterior part moves only slightly; the posterior part moves more freely.

SUMMARY

A man was seen in whom a paralysis of the medial and anterior portion of the right hemidiaphragm was present as a remainder of an old anterior poliomyelitis. Scoliosis to the contralateral side and considerable functional damage were found.

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THE ROENTGENOLOGICAL CHANGES IN THE ESOPHAGUS IN TUBERCULOUS MEDIASTINITIS*

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THE differential diagnosis between mediastinal tumor and mediastinal infection is often difficult for the roentgenologist as well as for the clinician. The only roentgenographic change may be widening of the mediastinal shadow without parenchymal lung disease. Any distinctive roentgenological sign which would aid in proving one diagnosis or the other would be of great value. Recently, examination of the barium-filled esophagus in addition to the usual chest roentgenograms has given promise of furnishing considerable differential aid. Five patients, all with active adult mediastinal tuberculosis, were studied in this manner, and in each case the value of esophageal study was confirmed.



FIG. 1. Tracing of anteroposterior esophageal spot roentgenograms in Cases I, II, III, IV and V.

CASE REPORTS

CASE 1.† (U-270125) *Purulent Tuberculous Mediastinitis.* (Proved by postmortem.)

A male, aged seventy-two, engineer, had had malaise with frequent chills and fever to 100°F. for six weeks. He had gradually become drowsy and lethargic. He had lost 50 pounds in weight. No definite diagnosis had been established. On admission he was acutely ill, drowsy, with a temperature of 100°F. and a leukocyte count of 18,600. The lungs contained a few basal râles and a patch of tubular breathing in the right scapular region. The spleen was enlarged.

Roentgenograms on admission showed a widened, slightly lobulated mediastinal shadow and partial collapse of the right middle lobe (Fig. 2). The barium-filled esophagus was displaced to the right above the aortic arch and to the left below it. The esophagus was flattened in the anteroposterior diameter, and the mucosal pattern at the junction of the middle and upper thirds was irregular. The spot roentgenograms were of poor quality and the examination was incomplete due to the patient's poor cooperation.

He died on the sixth hospital day.

Postmortem Examination. A 5 cm. ulcer of the esophagus was found just below the level of the carina. The ulcer had raised soft and necrotic edges; its base was gray and necrotic. A perforation from the base of the ulcer extended anteriorly into several markedly enlarged lymph nodes which surrounded the trachea, bronchi and esophagus. These nodes were also soft,

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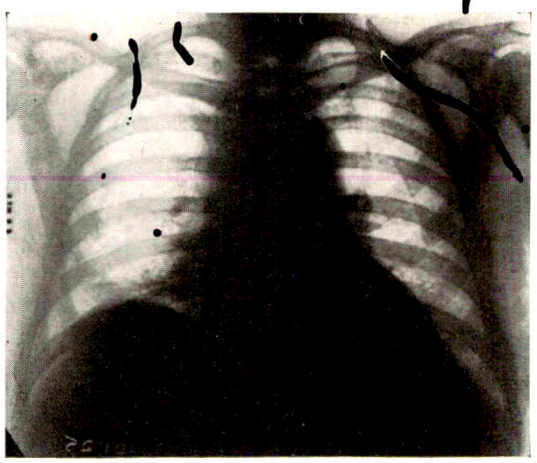


FIG. 2. Case 1. Purulent tuberculous mediastinitis. Widened mediastinal shadow and partial collapse of the right middle lobe.

gray and necrotic. The mediastinal tissues contained several small abscesses, and the septic process had extended through the fascial planes into the neck, with a small abscess at the lower pole of the thyroid gland. The gross anatomical diagnosis was carcinoma, but microscopic examination showed caseation, necrosis and typical tubercle formation with numerous tubercle bacilli. Tubercles were also present in the lungs, spleen and pericardium.

Comment. After six weeks of unexplained fever and chills, the patient showed mediastinal widening by roentgenogram. With this widening, the esophagus was flattened, displaced, and showed an irregular mucosal pattern. The clinical and roentgenological diagnoses were carcinoma. At postmortem examination, a tuberculous mediastinitis with ulceration into the esophagus was found.

CASE II. (U-336763) *Nonspecific Granulomatous and Caseating Mediastinitis.* (Proved by postmortem.)

In mid December, 1941, a boy, aged sixteen, developed headache, malaise and had a shaking chill. Shortly after this, a red papular and vesicular skin rash appeared and recurred several times. There was transient pain, redness and swelling of the knees.

On admission in January, 1942, the patient was pale with a multiform skin eruption. There was slight tenderness and swelling of both knees. His temperature ran between 100 and 102°F. Repeated leukocyte counts never rose

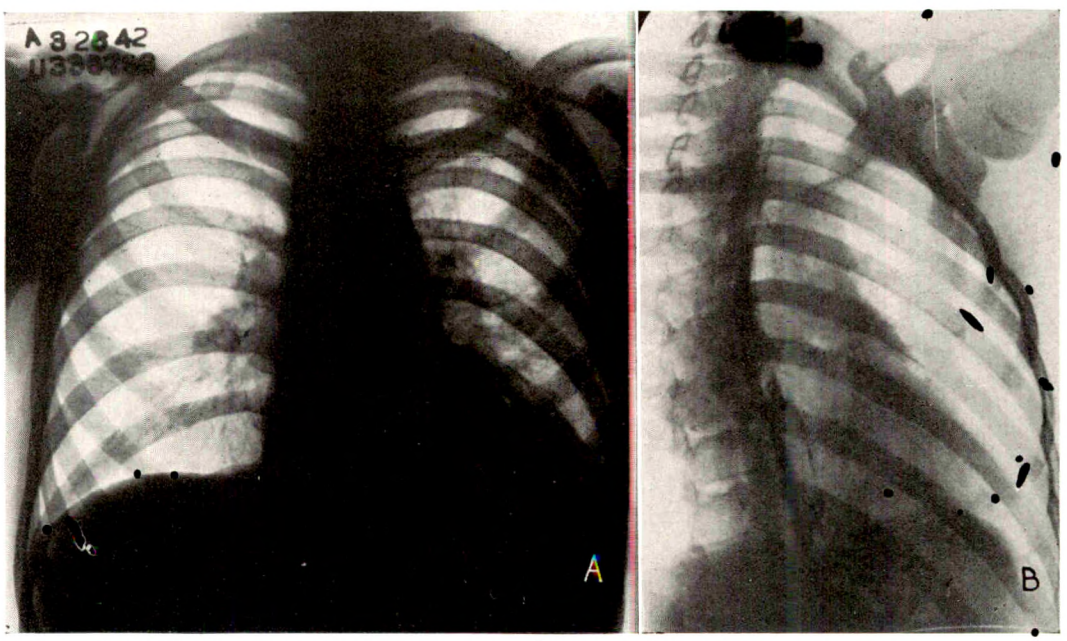


FIG. 3. Case II. Nonspecific granulomatous and caseating mediastinitis. *A*, enlarged hilar lymph nodes and density in the left lower lobe. *B*, oblique view of the barium-filled esophagus. The lower third shows irregularity in width and numerous projections from the wall.

above 5,400. Several clinical diagnoses were suggested.

Roentgenograms of the chest were negative in January. On January 23 he complained for the first time of a dull pain in the right lower anterior chest, increased by swallowing and deep breathing. Two days later an unproductive cough developed. One month after admission, roentgenograms of the chest (Fig. 3*A*) showed for the first time enlarged hilar glands and slightly increased density in the left lower lobe. At this time he raised a small amount of blood-tinged sputum.

Barium examination of the esophagus (Fig. 3*B* and 4) March 19–20, 1942: In the lower barium-filled esophagus for a length of 7 cm. there was marked irregularity of the wall with several projections (arrows). The width of the lumen was irregular. Normal mucosal folds passed through this area (Fig. 4*A*). A short, angulated, barium-filled fistula was demonstrated (Fig. 4*B*). These changes were interpreted as signifying peri-esophageal infection.

On March 26, the left lower lobe became completely consolidated. Four months after the onset of his illness he had a sudden hemoptysis and died.

Postmortem Examination. The superior and posterior portions of the mediastinum were filled with greatly enlarged lymph nodes which were matted together. On section, they were both firm and caseous. A small sinus ran from a diverticulum of the esophagus into a mass 4 cm. in diameter beneath the carina. The diverticulum was 1 cm., the sinus 2–3 mm. in diameter. There was a questionable second sinus into the left main bronchus. Both upper lobes were consolidated and contained several gray tubercles. There was an acute fibrinous pleuritis. The periaortic and retroperitoneal lymph nodes were similar to those in the mediastinum. The microscopic as well as the gross appearance was typical of tuberculosis. No tubercle bacilli could be found, however. The final pathologic diagnosis was a "nonspecific granulomatous and caseating process."

Comment. After three months of unexplained fever with a gradual, retrogressive course, and one month after the onset of substernal pain, barium examination of the esophagus localized the infection to the mediastinum, by demonstrating actual esophageal wall involvement. The roentgenographic changes were multiple adhesions to the esophageal wall, a short fistula into the mediastinum, and enlarged hilar shadows. Autopsy revealed a caseating and granulomatous mediastinitis and retroperitoneal adenitis.



FIG. 4. Case II. Spot roentgenograms. *A*, note the points of attachment of adhesions to the wall (arrows). *B*, with less filling, a fistula is seen. The mucosal pattern in the widened area is irregular.

CASE III.* (U-328726) *Tuberculous Bronchitis.* (Proved by biopsy.)

A male, aged thirty-seven, shoe-worker, suddenly became hoarse in February, 1941. In April, he developed a paroxysmal cough productive of black and green colored sputum, and slight exertional dyspnea. In November, there was a sudden onset of dysphagia with solid foods. On admission in December, 1941 (ten months after the onset of symptoms), the left vocal cord was paralyzed and the trachea was shifted slightly to the right. The temperature was normal; the leukocyte count was 8,100. The lung fields were clear by physical and roentgenological examination. Roentgenograms of the chest demonstrated a mass in the upper mediastinum just superior to the arch of the aorta.

* Case III was previously published as Case 28201, Case Records of the Massachusetts General Hospital, *New England J. Med.*, 1942, *226*, 825–828.

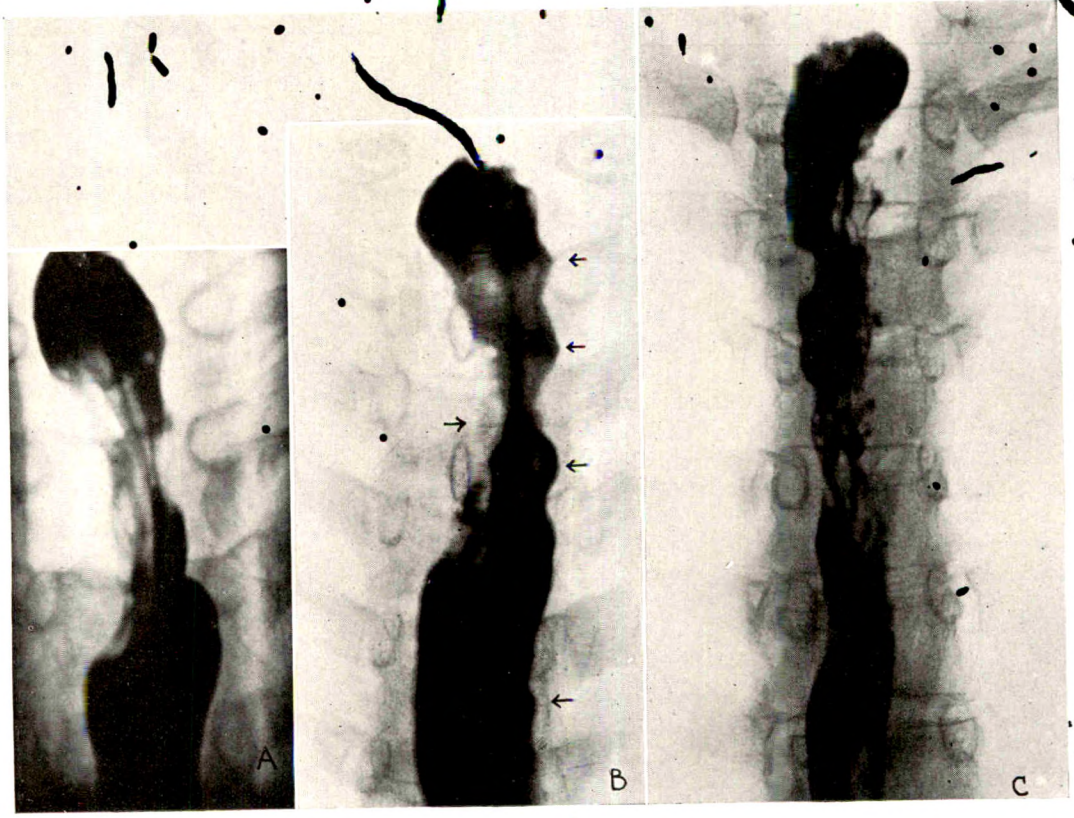


FIG. 5. Case III. Tuberculous bronchitis (spot roentgenograms). *A*, December 8, 1941. Narrowing of the esophagus with a large pressure defect from a mediastinal mass. *B*, January 1, 1942. Increase in length of the involved esophagus and numerous projections due to adhesions (arrows). *C*, April 26, 1942. Further extensions of the process and irregularity of the mucosal pattern.

A swallow of barium on December 8, 1941 (Fig. 5*A*), showed pressure on the esophagus from the mass in the upper mediastinum. The esophagus was narrowed over a length of 4 cm., most markedly in the anteroposterior diameter. The mucosa appeared destroyed. The roentgenological diagnosis was carcinoma. Three weeks later, the length of the involved segment had increased (Fig. 5*B*).

Bronchoscopy revealed an irregular reddening and outcropping of the left main bronchus, of the right upper lobe bronchus, and of the carina. Esophagoscopy showed no abnormality. The patient was given 1,200 roentgens at 200 kv. to the anterior and posterior mediastinum as a test dose for lymphoma; there was no change.

The first biopsy from the bronchial mucosa was reported as chronic inflammation; a second bronchial biopsy in February, 1942, showed tuberculosis.

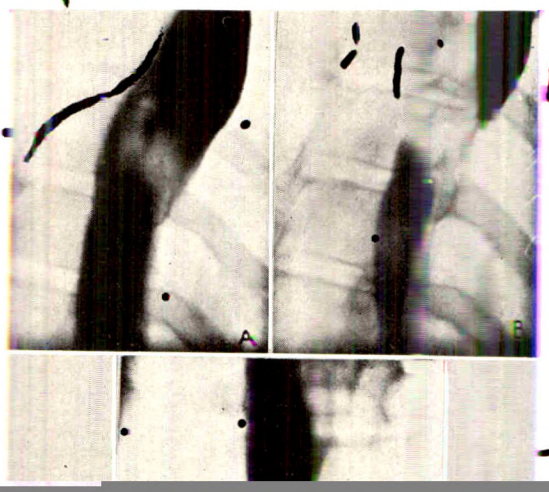
In April, 1942 (Fig. 6), the lung fields were still clear, and the mass in the upper mediastinum had not changed in size; it was compressing the lower trachea. The length of the involved esophagus had again increased (Fig. 5*C*). The mucosa pattern was irregular and broken up. Several projections from the wall were seen on the spot roentgenograms and were interpreted as due to adhesions.

The patient was discharged to a tuberculosis sanatorium.

Comment. A proved case of tuberculous bronchitis without peripheral lung disease showed a mediastinal mass, with pressure on and adhesions to the esophagus in the region of the mass. The mucosal pattern was irregular in the involved segment, but direct visualization of the esophageal mucosa showed no abnormality. Figure 5 shows the gradual lengthening of the involved area over a period of five months.

CASE IV. (U-27439) *Acute Tuberculous Adenitis.* (Proved by biopsy.) A woman, aged thirty-four, five weeks before admission had a chill, followed by several less severe chills. The temperature rose to 100 and 102° F. after each chill. There was mild pain in the knees. Examination in April, 1942, showed a pale woman with enlarged cervical and epitrochlear glands. The





fect are seen sharp and cleanly cut off. This was interpreted as indicating intimate adherence of the esophagus to the bronchus.

Biopsy of a left supraclavicular gland showed active tuberculosis.

The patient improved steadily on bed rest and was discharged home. Six months later she was fully active and had gained 4 pounds in weight. There was no palpable adenopathy. The mediastinal shadow was not enlarged. The barium-filled esophagus (Fig. 8*C*) showed the adherence to the bronchus and, in addition, below the bronchus two small, sharp, pointed projections. The lumen of the esophagus varied slightly in size for 3 inches below the crossing bronchus without definite filling defect. Peristalsis was sluggish in this region.

Comment. A young woman who had had chills and a low grade fever for five weeks showed enlarged hilar lymph nodes without parenchymal lung disease in the roentgenograms. Involvement of the esophagus in the inflammatory process was shown by the intimate adherence of the esophagus to the left main bronchus. Six months later, the esophagus showed, in addition, two adhesions just below the bronchus. Biopsy of a supraclavicular gland showed active tuberculosis.

CASE V. (U-264903) *Active Pulmonary Tuberculosis and Mediastinal Adenitis.* (Proved by positive sputum.)

An aching sensation over the left lower anterior chest, not increased by respiration, was the chief complaint of this patient, male, aged sixty-eight. This had been present for six weeks, and for one month he had had an unproductive and paroxysmal cough. There had been increasing dyspnea, weakness, and at times a choking sensation. Examination showed a weak, elderly man with diminished breath sounds in the left posterior chest without râles. The clinical diagnosis was cancer of the lung.

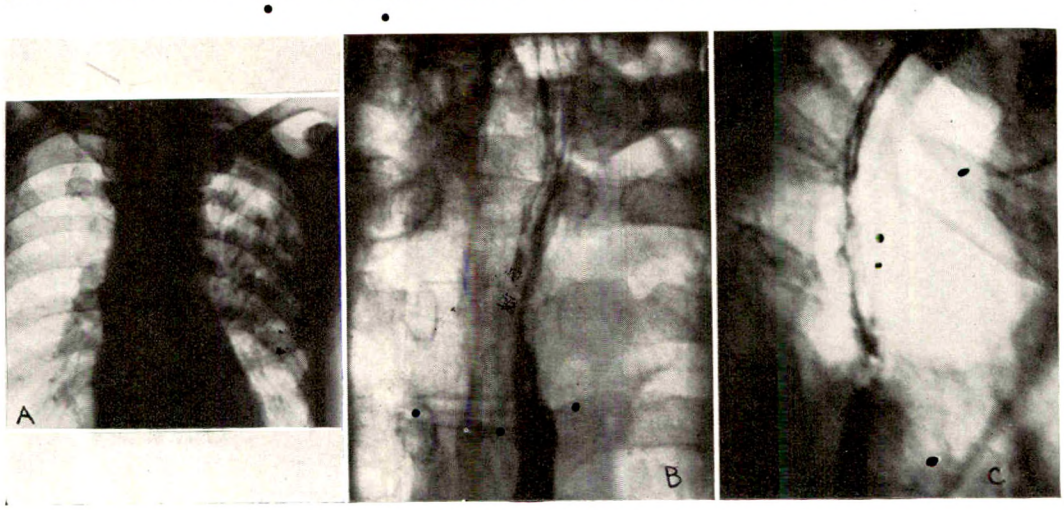


FIG. 9. Case v. Active pulmonary tuberculosis. *A*, mottled density in the left lung fields. Enlarged lymph node beneath the aortic arch. *B*, barium-coated esophagus shows pressure defect due to enlarged glands, the irregularity of mucosal pattern and fine waviness of outline. Posteroanterior view. *C*, lateral spot roentgenogram.

Roentgenologic Examination. There was mottled dullness with calcification in the mid left lung field (Fig. 9*A*). A large gland (arrows) was seen just beneath the aorta, pushing the left main bronchus downwards and the trachea to the right. Above the arch, the trachea was deviated to the left.

Barium swallow (Fig. 9*B* and 9*C*) showed, at the level of the enlarged subaortic gland, a segment of the esophagus about 5 cm. in length which differed from the esophagus above and below. There was a fine waviness of the wall and slight irregularity of the mucosal pattern. There were two small pressure defects on the left side, where small lobulations of the enlarged gland appeared to press against the esophagus.

Bronchoscopy showed marked pressure on the left main bronchus without mucosal in-

involvement. The gastric contents and concentrated sputum contained many tubercle bacilli.

Comment. A case of moderately advanced pulmonary tuberculosis with enlarged mediastinal glands showed a short segment of the esophagus involved at the level of the enlarged glands. There were multiple fine adhesions, some irregularity of the mucosal pattern and two small pressure defects on the esophagus.

PATHOLOGY

Tuberculous mediastinitis usually develops from infection in the tracheobronchial lymph nodes, and infrequently from rupture of a tuberculous focus in the spine, sternum, pericardial or pleural cavities. Though tuberculous mediastinitis is a rare disease, the types of tuberculosis which may lead to it are several. Active pulmonary tuberculosis is the most frequent source. The breaking down, or reactivation, of an old calcified or scarred mediastinal gland, when no active pulmonary tuberculosis is present, may be the cause in older patients. Tuberculous tracheobronchitis rarely gives rise to mediastinitis.[5,12] There may be an acute generalized tuberculous adenitis which, though clearly seen in the mediastinum, has been found also in the cervical and epitrochlear regions.[15]

The tracheobronchial lymph nodes, when infected with the tubercle bacillus, enlarge and coalesce into inflammatory masses.[11] The loose areolar tissue close to the nodes may also become infected, and numerous mediastinal abscesses form. The peri-adenitis which develops, however, may not be purulent but chiefly reactive, and result in multiple adhesions and scars. The firm union of the nodes to one another is due to this peri-adenitis and later scarring. At postmortem examination there is frequently found in the adult a scarred or calcified node, or mass of nodes, firmly bound by adhesions to the trachea or bronchus.

Normally the mediastinal connective tissue stretches loosely from the lymph nodes to the trachea, bronchi, great vessels and esophagus. After infection of this tissue, the resulting adhesions and scars may pull upon and stretch the esophagus, or a scar or part of an inflammatory mass may wholly or partially encircle the esophagus and lead to partial stenosis or a small area of rigidity in its wall. The enlarged lymph nodes may press against the wall or displace the esophagus from its usual course. The infection may spread to the esophagus; with tubercle formation in the wall, ulceration and final rupture into the esophagus may occur.

The above description is of an *active* tuberculous infection in the mediastinum. In the adolescent and young adult the tracheobronchial lymph nodes usually enlarge and later scar and calcify without a generalized mediastinal reaction. The scarring may be marked at one point, however, and may involve a localized point in the esophagus, at which point the scar or adhesions may pull upon the esophageal wall and result in a traction diverticulum. Fischer[7] described a firm adhesion running from a group of lymph nodes to the apex of the diverticulum. He cites Beverman's 60 cases of traction diverticula, 51 of which showed inflammatory changes and scarring in the adjacent lymph nodes. Seventy-three per cent of these changes were tuberculous. Helm[4] described and illustrated with a tracing a traction diverticulum over "an indurated tuberculous process of the inferior tracheobronchial lymph nodes."

ROENTGENOLOGICAL FINDINGS IN TUBERCULOUS MEDIASTINITIS WITH PARTICULAR REFERENCE TO THE ESOPHAGUS

Several roentgenological signs of active tuberculous mediastinitis have been described. Of these the classical sign is widening of the mediastinal shadow to the right and to the left. The association of mediastinal widening with tuberculosis in the lungs, pericardium and vertebrae has been emphasized. Wessler[15] described for the first time enlargement of the hilar and mediastinal glands without pulmonary disease as a manifestation of active tuberculosis in 3 young persons. He called the process in-

trathoracic tuberculous lymphoma" because of the lobulated mediastinal shadow which simulated tumor. Kornblum and Osmond[10] and Keefer[6,8] described similar cases.

The effect of active tuberculous mediastinitis on the esophagus has not been mentioned frequently in the literature. Displacement or deviation of the esophagus from its usual course has been described.[8,10] Keefer presented a case in which the esophagus was compressed with obstruction to the passage of barium. Kornblum and Osmond[10] found in 1 case an area of constriction with dilatation above and traction diverticula. Belden[1] noted slowness in the passage of barium through the portion of the esophagus adjacent to the infection. I was not able to find a recorded case with actual involvement of the inside of the esophagus by mediastinal tuberculosis. In all 5 of our cases which were seen in the course of one year, changes in the roentgenologic appearance of the esophagus were observed, and in 2 cases actual involvement of the esophageal mucosa was present. Figure 1 shows the significant changes in each case. The black and white tracing was made from spot roentgenograms taken during roentgenoscopy in the posteroanterior projection. The segment of the esophagus which was involved was adjacent to the enlarged glands or inflammatory mass.

Displacement of the esophagus from its usual course by soft tissue masses and pressure defects on the barium-filled lumen were seen in 3 cases. The esophageal mucosa was involved in 2 cases and showed roentgenologically as an irregularity of the normal mucosal pattern. In Case 1, postmortem examination showed a large tuberculous ulcer of the esophagus. In Case II, a tuberculous sinus ran from a small diverticulum of the esophagus into caseating mediastinal glands. In Case III, the irregularity of the mucosal pattern was so marked that ulcerations of a carcinoma were simulated. Esophagoscopy, however, showed no abnormality of the mucosa. The irregularity may be due to the folding and kinking of the wall and mucosa by scarring and very small inflammatory masses.

The esophageal wall in the involved area varied in width in 4 cases when the lumen was filled with barium. In some places the lumen was narrower than usual; in others, it was stretched out. In 1 case the lumen, when stretched out, failed to collapse upon itself normally during contraction. The stenosis is due to pressure of inflammatory masses or scarring; the stretching out is probably the result of adhesions. A fistula was demonstrated in 1 case and was found at postmortem in 2 other cases.

In Case IV (tuberculous adenitis), intimate adherence of the esophagus to the left main bronchus was the only indication of peri-esophageal infection. The spot roentgenograms (Fig. 8) show the oblique cylindrical pressure defect of the bronchus. The significant change, however, is the sharp edge of the barium column where the esophagus meets the bronchus. This is seen only when the esophagus is partially filled (Fig. 8*B*) and is obscured by complete filling (Fig. 8*A*). This sharp definition was produced experimentally in the esophagus by Schatzki and Hawes[14] with an extrinsic object, when the object was *firmly* attached to the esophageal wall. That the esophagus was closely bound to the bronchus by adhesions was confirmed by the demonstration later of adhesions below the bronchus.

The unusual finding in 4 of these cases was a number of projections, appearing either as points or rounded waves, from the involved segment of the barium-filled esophagus. In Case V (Fig. 9), the projections were so numerous that a fine, wave-like outline was given to the esophagus. These projections were not transitory. If in one spot roentgenogram a peristaltic wave happens to be photographed, a rounded projection of the wave will be seen, but in subsequent roentgenograms, taken over a period of several minutes to hours, the same wave is not seen. In our cases, however, the projections were always in the same position from film to film, and after a period of several hours when the patient was examined in the same position. The size and shape of the projections in the in-

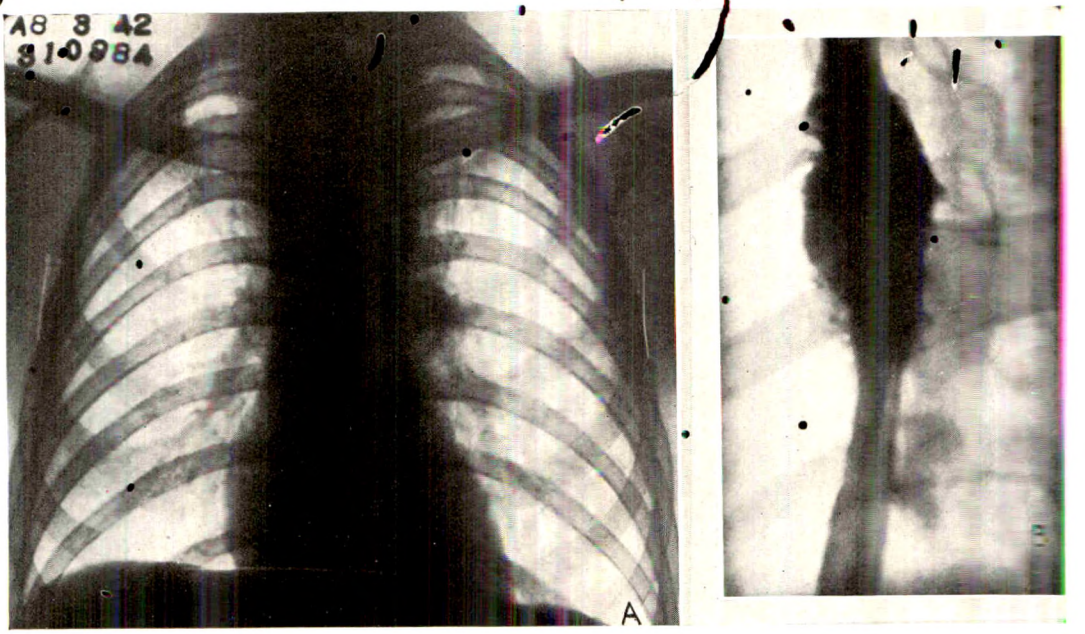


FIG. 10. A case of fibrous mediastinitis and streptococcal paravertebral abscess. *A*, widened superior mediastinal shadow. *B*, spot roentgenogram of the barium-filled esophagus at a level just above the aortic arch shows widening of the lumen and several adhesions.

volved segment, on the contrary, could be varied by the degree of barium filling.

The pathologic explanation of these projections is probably related to the scarring and adhesion formation in the peri-esophageal tissues. In the past, roentgenological changes in the esophagus which are believed to be due to adhesions have been described.

The best known example of the effect of an adhesion on the esophagus is the traction diverticulum, which is frequently seen in adults roentgenoscopically. Schatzki[13] reported a diverticulum with a calcified gland close to the fundus and another within a mass of actinomycotic inflammatory tissue.

Fleischner[3] described another type of diverticulum, a transitory outpouching of the esophageal wall, visible only in the phase between complete barium filling and contraction of the lumen. He attributed its formation to absent contraction at one point in the esophageal wall due to abnormal adherence of this point to the peri-esophageal tissues.

Schatzki[13] called attention to a peculiar phenomenon which is seen occasionally during the contraction of the esophagus. Following deglutition an otherwise smooth esophagus may suddenly show multiple toothlike projections. The phenomenon was called Kräuselung or "curling." These irregularities, although inconstantly present, seem to recur in identical places in a given case. This finding, as well as the fact that "curling" is commonly associated with characteristic adhesion diverticula, has led to the assumption that it is probably produced by localized adhesions of the esophagus. The entire picture cannot be explained on this basis, and it is possible that functional changes may occur secondary to adhesions.

In a case of streptococcal paravertebral abscess which we examined, similar changes in the esophagus were demonstrated.

A physician, aged twenty-eight, had had intermittent backache for three years; for one year a paravertebral mass had been seen along the thoracic vertebrae. In August, 1942 (Fig. 10*A*), there was, besides the paravertebral mass, widening of the superior mediastinum. Barium examination of the esophagus (Fig. 10*B*) showed the small projections from the wall of

the barium-filled esophagus which were interpreted as adhesions. The lumen was irregular in width and failed to collapse upon itself on contraction of the remainder of the esophagus. The lung fields were clear.

Aspiration of the paravertebral mass drew out non-hemolytic streptococci.

The patient became anuric and died.

Postmortem Examination. The posterior and superior mediastinum contained a large amount of firm fibrous tissue from the level of the eighth to the tenth dorsal vertebrae. This fibrous tissue was 0.5–2.0 cm. in thickness, and at the level of the fourth and fifth dorsal vertebrae it encircled the esophagus and was adherent to the posterior tracheal wall. In one place a firm, fibrous band constricted the lumen slightly. Posterior to this thick fibrous mediastinal tissue mass, there was a shallow paravertebral abscess which did not communicate with the major mediastinal cavity.

Comment. The segment of esophagus shown roentgenologically as involved by adhesions was found at postmortem examination to be surrounded by thick fibrous tissue which narrowed and pulled upon the esophagus.

Projections, as discussed above, are not physiological, but are due to the peri-esophageal tissue changes, and are analogous in etiology to traction diverticula, Fleischner diverticula, and probably "curling." The finding of these projections in the esophagus of a young person is of considerable differential diagnostic importance. In Case II, their discovery localized the cause of obscure fever to the mediastinum when other roentgenologic and clinical studies had failed. In association with signs of active infection and within mediastinal widening, their demonstration lends considerable weight to the diagnosis of mediastinal infection rather than tumor. One should ascertain that operation on the mediastinum or lungs has not been performed, as such operative trauma may produce numerous mediastinal adhesions.

SUMMARY

1. The roentgenologic changes in the esophagus in five cases of active tuberculous mediastinitis are described. The appearance of the esophagus had been changed by pressure, by adhesions, and by actual involvement of its mucosal surface.

2. The importance of demonstrating esophageal adhesions in the differential diagnosis between mediastinal tumor and infection is emphasized.

I wish to express my appreciation to Dr. A. O. Hampton, who suggested this study, and to Dr. Richard Schatzki, who offered numerous constructive comments and criticism of the manuscript.

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VOL. 51, NO. 5

CORRELATION OF ROENTGENOLOGIC AND GASTROSCOPIC EXAMINATIONS

FROM THE STANDPOINT OF THE GASTROENTEROLOGIST*

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IT IS essential that we pause from time to time to take inventory of our diagnostic and therapeutic methods. Too often when something new is offered we are likely to accept it carelessly, being a little too enthusiastic about its virtues and forgetful of its limitations. Ten years ago the flexible gastroscope was introduced and for a few years enjoyed the ardor of its more zealous disciples. Today, however, gastroscopy is in a period of re-evaluation. This study, which is an evaluation of our experience with gastroscopy, is not an attempt to establish the relative superiority of gastroscopy over roentgenology. All students of the subject agree that the methods are supplemental and complemental, not competitive. Furthermore, evidence is accumulating to show that the percentage of accurate diagnoses is greater if both methods are used than if either method is used alone.

In order to determine the value of gastroscopy from the standpoint of the gastroenterologist, we reviewed 938 consecutive examinations made on 842 patients.[1] In this evaluation of gastroscopy let us consider (1) the gastroscopic contributions of primary value, (2) the contributions of secondary value, (3) the failures to contribute information to cases, (4) the reasons for the failures, and (5) a comparison of the roentgenologic and gastroscopic diagnoses in a selected group.

Primary Value of Gastroscopy. Before classifying gastroscopy as making a primary contribution to a case, the gastroscopist had to add an entirely new diagnosis or definitely establish a doubtful diagnosis, thereby materially altering the treatment and prognosis from what it might otherwise have been. With this criterion we found 217 patients, or 25.6 per cent of the 842 patients examined, in whom the gastroscopic examination was of major value as compared to the roentgenologic. In 150 patients, or 17.7 per cent, the diagnosis was established by gastroscopy when other methods had failed, the roentgenologic examination being negative. Most of these were cases of chronic gastritis, but it

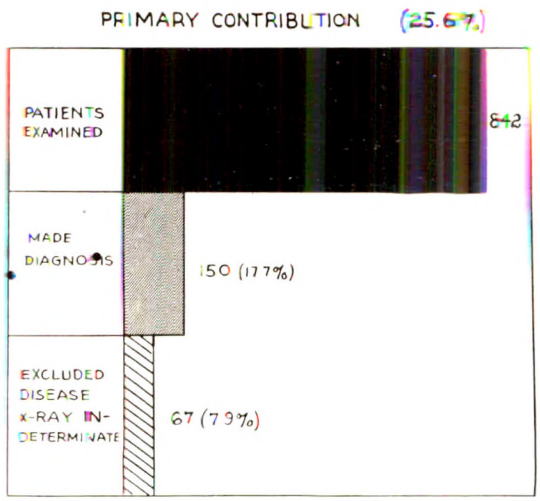


FIG. 1. Gastroscopic diagnosis of major value—a primary contribution.

is significant that there were 10 gastric ulcers not revealed by the roentgenologic examination. It is as important to exclude organic disease as it is to establish its presence. In 67 patients, or 7.9 per cent of all examined, the roentgenologist suspected gastric ulcer or cancer. In each instance the gastroscopist had adequate and satisfactory visualization of the area in question and could state definitely that no organic disease was present, thereby adding a major contribution to the case.

* Presented at the Forty-third Annual Meeting, American Roentgen Ray Society, Chicago, Ill., Sept. 15–18, 1942.

Secondary Value of Gastroscopy. Gastroscopy was of secondary importance as compared to the roentgenologic examination in 55.4 per cent (Fig. 2). We believed that confirmatory evidence was of some value although not of primary significance. The gastroscopic confirmation of a roentgenologically normal stomach in 238 cases, 28.3

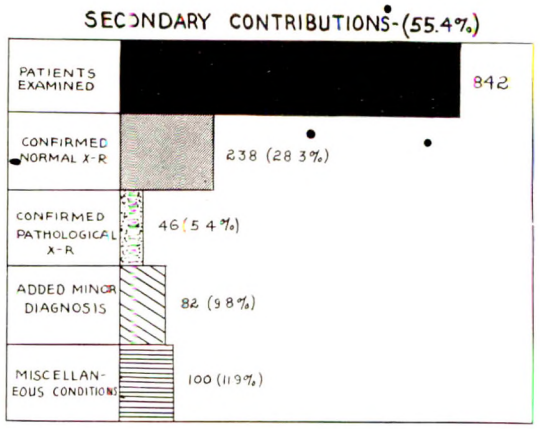


FIG. 2. Gastroscopic diagnosis of secondary importance compared to major diagnosis.

per cent, definitely helped to evaluate these particular cases. Likewise, the confirmation of a pathological finding was both of comfort and of value to the clinician, because the percentage of accurate diagnoses is greater when the gastroscopic and roentgenologic diagnoses are in agreement. The remaining 182 cases might be better classified as being of academic interest rather than of secondary value. In 82 of these cases the presence of chronic gastritis with duodenal ulcer was demonstrated. The other 100 cases included such diagnoses as atrophic gastritis with pernicious anemia, pigment spots, and so forth.

Valueless Gastroscopic Examinations. Whereas gastroscopy was of major value in 25.6 per cent and of secondary value in 55.4 per cent of the cases examined, it was of no value in 19 per cent. As shown in Figure 3, the largest number of unsuccessful examinations occurred because the gastroscopist failed to visualize a lesion or an area where the roentgenologist suspected or demonstrated a lesion. There were 58 such instances. The next largest number was due to anatomical difficulties such as esophageal spasm, acute angulation of the esophagus preventing entrance of the gastroscope into the stomach, or angulation of the instrument beyond its useful range from other causes. The indeterminate and wrong diagnoses ranked third with 33 cases. Unusually large blind spots were present in 13 examinations. In 9 instances the patient's cooperation was so poor that the examination was incomplete. Twenty-four stomachs were examined postoperatively, and 5 times the stoma was not satisfactorily seen. These should be properly classified as "area in question not seen," but for statistical purposes the stomachs examined postoperatively were considered separately. In 4 instances the instrument did not function properly. Twice reactions to the medications used in the preparation of the patient and once a gastric hemorrhage prevented carrying out a gastroscopic examination for which the patient had been prepared.

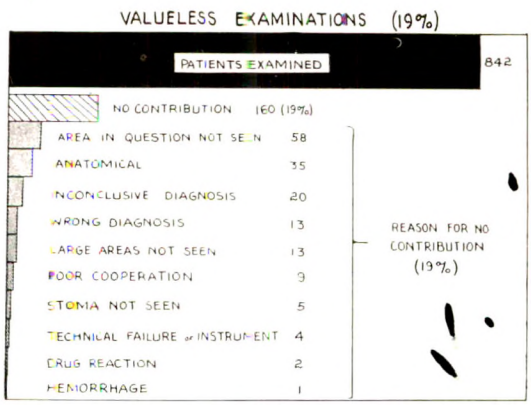


FIG. 3. Gastroscopic examination of no value. Reasons for no contribution to case history.

Limitations of Gastroscopy. Of the limitations of gastroscopy, the technical and optical problems are probably the least well known by the clinician. Of all endoscopes the gastroscope is longest and traverses the most tortuous and difficult route. The Schindler-Cameron gastroscope contains 54 lenses. If for anatomical reasons the in-

strument is flexed beyond 42°, the image is not transmitted from one lens to the next. Severe temporary esophageal or gastric spasm may render the examination incomplete. The shape and size of the stomach is constantly changing because of (1) respiratory excursions, (2) peristalsis, (3) cardiac and aortic pulsations, (4) passive distention with air, or (5) collapse. The gastroscopist, therefore, sees a "moving picture," and what may be seen at one movement cannot be visualized again. Although the roentgenologist may experience difficulties due to body build and position of the stomach, it is not comparable with the gastroscopist's problem. Furthermore, the roentgenologic examination may be easily prolonged or repeated almost at will. The gastroscopist can inspect the mucosa only, while the roentgenologist sees the entire stomach wall. But more important than these problems are the gastroscopic blind spots. Certain areas are never seen or are so seldom partially visualized that they may be considered as "constant blind areas" (Fig. 4). These include an area at the tip of the instrument,

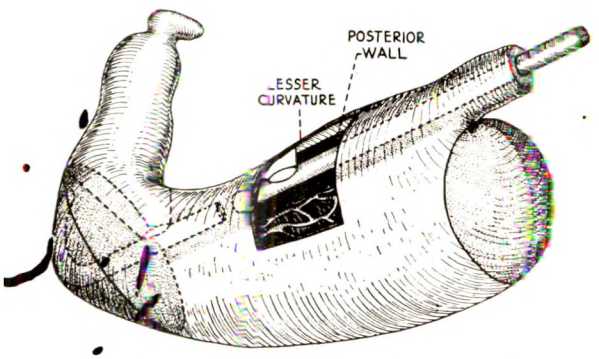


FIG. 4. Constant blind areas. Strip on posterior wall of body may actually be larger than indicated.

extending over the greater curvature; the lower anterior and posterior walls opposite the incisura angularis; the posterior wall of the body immediately adjacent to the instrument; the extreme upper portions of the lesser curvature; and a large portion of the fornix. Occasionally, one may see a portion of these areas, especially the posterior wall of the body, but for practical purposes they are "constant blind areas." In addition to these areas there are others which are seen with variable frequency. These "inconstant blind areas" include the pylorus, lesser curvature of the antrum, usually large amounts of the posterior wall of the body, and at times other areas depending on anatomical factors.

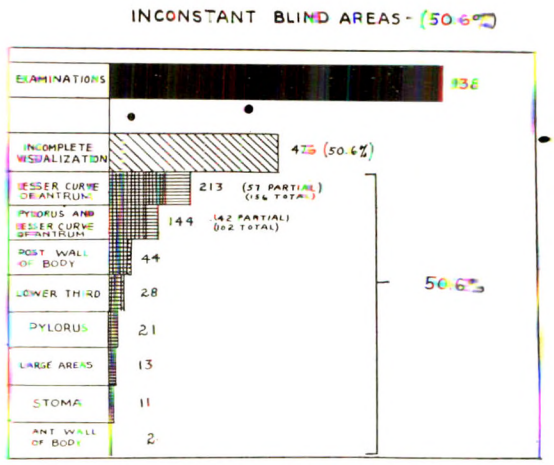


FIG. 5. Inconstant or variable blind areas.

Since the inability to visualize the area in question occurred so frequently, the gastroscopic examinations were analyzed according to the variable or inconstant blind areas (Fig. 5). Four hundred and seventy-six of the 938 examinations were to some degree incomplete, although in only 118 cases was the examination totally unsatisfactory because of technical reasons. Failure to visualize a part or all of either the pylorus or lesser curvature of the antrum in 378 patients accounted for 79.4 per cent of the 476 incomplete examinations. The remaining cases included those in which an unusually large amount of the posterior wall, the lower third of the stomach, the stoma, the anterior wall, or a combination of these large areas were not satisfactorily visualized.

One point here needs emphasis, namely, the matter of multiple gastroscopic examinations in the same patient. An incomplete or indeterminate gastroscopic exam-

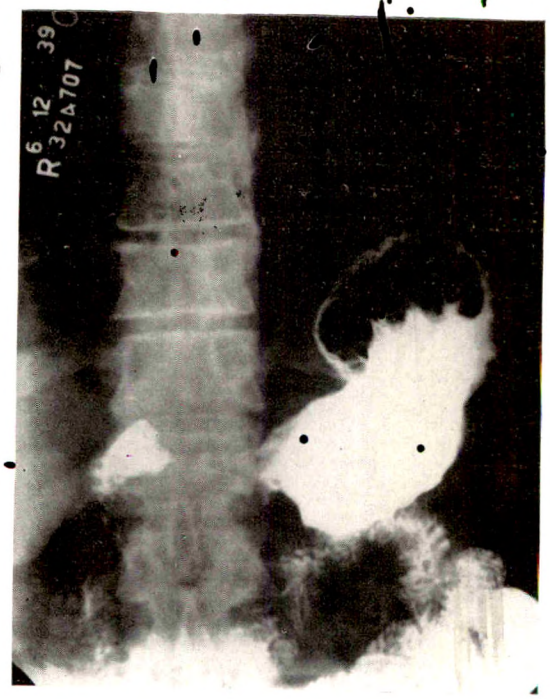


FIG. 6. Antral deformity was persistent in all views and during roentgenoscopy. Interpreted as cancer.

ination does not mean that all subsequent examinations will be unsatisfactory any more than the same may be said of the

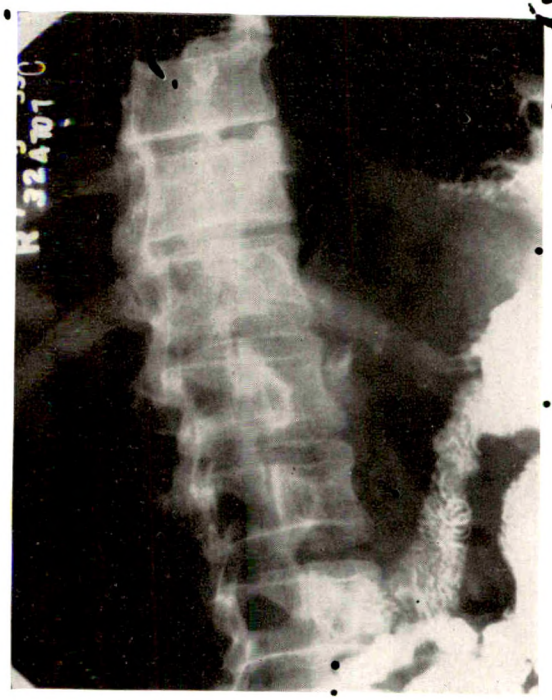


FIG. 8. Second roentgenologic examination three weeks later. Antral deformity persistent at roentgenoscopy and unable to keep barium in antrum for satisfactory roentgenograms.



FIG. 7. Gastroscopic view. Findings indeterminate. Unable to state whether orifice with bubbles regurgitating represented pylorus or proximal end of antral deformity.

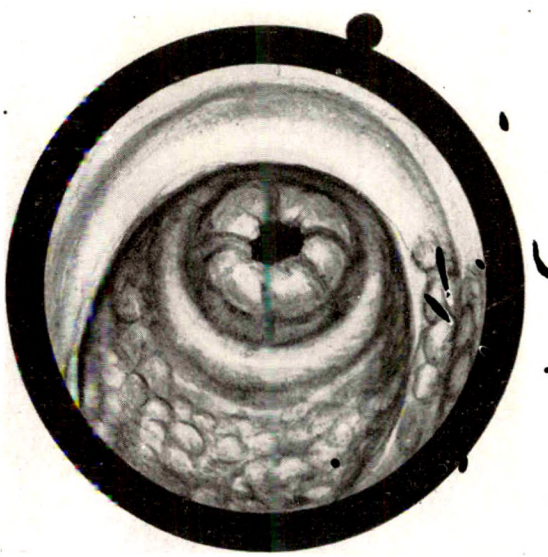


FIG. 9. Second gastroscopic examination showing pylorus in partial contraction with approaching peristaltic wave. Typical changes of hypertrophic gastritis.

roentgenologic examination. It has been our observation that the clinician may be willing to request one gastroscopic examination, but it is uncommon that subsequent examinations are requested although the roentgenologic examination may be repeated many times. We believe this is due to lack of faith and knowledge or a to misunderstanding of some of the limitations of gastroscopy. As a matter of fact, "progress" or repeated gastroscopic examinations should be used. In certain cases we have learned more from "progress" gastroscopic examinations than from the "progress" roentgenologic study. One case which is typical of several in our experience will illustrate the point.

A man aged forty-nine consulted us because of (1) epigastric pain, (2) substernal pain, and (3) bowel distress. After complete investigation diagnoses were made of arterial hypertension with coronary artery disease and angina pectoris, chronic cholecystitis, cholelithiasis, and diverticulosis of the colon. In addition, roentgenologic examination revealed a persistent annular prepyloric defect (Fig. 6) interpreted as cancer. At the first gastroscopic examination (Fig. 7) we were uncertain and indeterminate about our diagnosis. Three weeks later the roentgenologic findings were essentially the same (Fig. 8), but the gastroscopic picture was entirely different (Fig. 9). All parts were well seen, and there was no doubt about the diagnosis of hypertrophic gastritis. Today, two years later, this man is subjectively much improved and objectively no worse.

TABLE I

COMPARISON OF FIRST ROENTGENOLOGIC AND GASTROSCOPIC EXAMINATIONS

	Normal	Hypertrophic Gastritis with Normal or Hypertrophic Rugae	Benign Tumor or Polyp	Benign Ulcer	Cancer	Gastritis with X-Ray Ca or Ulcer	Stoma Ulcer	
Diagnosis in agreement	43 (4)	39 (6)		9 (13)	18 (16)			[illegible] [illegible]
Gastro-Correct X-Ray Incorrect Or Indeterminate	4		1 (1)	5	7 (4)	6 (1)		23 (5)
X-Ray Correct Gastro-Incorrect Or Indeterminate			1 (1)	9	(4) 4		1	
Both Incorrect	1			3 (3)	5 (5)			9 (3)
Both Indeterminate							14	14

* Figures in parenthesis indicate diagnoses proved by operation or autopsy.

Comparison of Roentgenologic and Gastroscopic Diagnosis. In the foregoing discussion gastroscopy has been considered in relation to roentgenology and the patient. However, in order to compare the accuracy of the roentgenologic and gastroscopic diagnosis, we selected 170 patients (Table 1) who had been observed for one year or longer, or in whom operations or autopsy had established the true diagnosis. As the gastroscopic and roentgenologic examinations were done within a few days of each other, they were comparable. Only those gastroscopic examinations in which areas

except the constant blind areas were visualized were accepted for study. Furthermore, since the first examination is so important and largely decides the clinician's choice of therapy, only the first examinations were considered. There were 109 cases, or 64.1 per cent, in which both the roentgenologist and gastroscopist were in agreement. Of these, 43 stomachs were normal, 9 had benign ulcer, 18 had cancer, and 39 had chronic gastritis with a roentgenologic report of either a normal stomach or with hypertrophic rugae. The latter group were classified as being in agreement because our roentgenologists believe it is chiefly the gastroscopist's prerogative to make a diagnosis of chronic gastritis.

In 15 cases the roentgenologist's diagnosis in the first examination was correct, while the gastroscopist's diagnosis was either indeterminate or incorrect. Nine of these cases were benign ulcer, 1 of which was an indeterminate diagnosis and 8 of which could not be seen gastroscopically, 4 cancer, 1 benign tumor, and 1 gastroenteric stomal ulcer.

In 23 patients the gastroscopist first made the correct diagnosis. It should be emphasized that a diagnosis was considered as indeterminate if the examiner qualified the diagnosis in any way, even though in his own mind he definitely favored a single unqualified diagnosis. Diagnoses such as "ulcer, probably benign but malignancy not definitely excluded" made by either the gastroscopist or roentgenologist were classified as indeterminate. If a lesion seen by one method was not seen by the other, it was an indeterminate examination for the latter method. In this group of 23 patients there were 4 gastroscopically normal cases in which the roentgenologist suspected a lesion. There were 6 cases of chronic gastritis causing a deformity which at first was suspected as being due to cancer or ulcer. There were 5 cases of benign ulcer, 2 of which were not seen roentgenographically, 7 of cancer, and 1 of benign tumor with an indeterminate roentgen report.

These three groups account for 147 of the 170 cases, or 86.4 per cent, in which both or one of the examiners made the correct diagnosis. In contrast to this there were only 9 instances, or 5.3 per cent, in which both the roentgenologist and gastroscopist were incorrect, and 14 cases in which both were indeterminate.

CONCLUSIONS AND SUMMARY

On the basis of our review of 938 gastroscopic examinations in 842 patients, we believe gastroscopy has a definite but limited diagnostic value. Certain cases cannot be considered as having been adequately and properly studied unless a gastroscopic examination has been done. From the standpoint of the gastroenterologist, these include (1) cases in which the roentgen findings are negative, but in which upper gastrointestinal disease is still suspected; (2) cases with indeterminate or inconsistent roentgenologic findings; (3) gastric ulcer especially while being treated; and (4) cancer except in advanced, terminal, or obvious cases.

It was our experience that gastroscopy was of primary or major value in 25.6 per cent of all cases examined. The primary value consisted of establishing a diagnosis which had not been made by other means or definitely making a diagnosis which by other means was indeterminate or incorrect. There were 17.7 per cent with negative roentenologic examinations in which gastroscopy revealed pathema (140 cases of gastritis and 10 cases of ulcer). In 7.9 per cent the roentgenologist suspected pathema which was excluded by gastroscopic examination.

It was also our experience that gastroscopy was of no value in 19 per cent of the cases. The chief cause of failure was the gastroscopist's inability to visualize certain areas where a lesion had been demonstrated or was suspected by the roentgenologist. Incorrect or misleading diagnoses constituted only 3.9 per cent of all cases examined.

A comparison of roentgenologic and gastroscopic diagnoses in a series of proved

diagnoses revealed that while the roentgenologic examination was still the most valuable single examination, the gastroscopist was no more likely to make the correct diagnosis or to err in judgment than the roentgenologist. Likewise, in the matter of differentiating between a malignant and benign lesion, the gastroscopist is not superior to the roentgenologist. In 60 instances of cancer or benign ulcer both examinations were in agreement in 27 cases. The gastroscopist was indeterminate or incorrect 13 times, the roentgenologist 12 times, and both were incorrect in 8 instances. Obviously, then, the proper procedure to follow when the roentgenologic and gastroscopic findings are not in agreement is further study and observation of the case utilizing both "progress" roentgenologic and gastroscopic examinations.

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DISCUSSION

DR. FREDERICK E. TEMPLETON, Chicago, Ill. For the past eight years I have worked in close association with Dr. Rudolph Schindler in comparing roentgen examinations with gastroscopy. From time to time we have been on opposite ends of the scale in our interpretation of these findings. Some of our reports, of course, are recorded in the literature. It is therefore very gratifying to me that the paper of Dr. Renshaw bears out my own opinions.

There are a few points, I believe, that should be emphasized. One is the identification and differentiation of chronic gastritis. The only gastritis we can identify is the hypertrophic form. In our experience this has included a very small number of cases of the hypertrophic gastritis, which the gastroscopist reported. The differentiation between a benign infiltrating lesion, such as hypertrophic gastritis, and an infiltrating cancer is extremely difficult and usually cannot be made with any degree of assurance by roentgenologic or even gross pathologic examination. The microscopic examination is the only sure method.

The gastroscopist, of course, diagnoses gastritis with far greater frequency than we roentgenologists, but I should like to ask Dr. Renshaw how many of these cases of gastritis are really of clinical significance.

There are a significant number of ulcers which we, as roentgenologists, cannot see. The existence of some of the ulcers has been demonstrated by pathologic examination. Most of the ulcers we do not see are the shallow forms. I sometimes wonder whether every white spot seen on the mucosa by the gastroscopist can be differentiated from a little collection of mucus. I know that Dr. Schindler has experienced difficulty making the differentiation. I should like to ask Dr. Renshaw how sure he can be or how many times he can be certain that some little area which looks like an ulcer is really an ulcer.

We all know that there are certain types of ulcerating carcinomas that we cannot differentiate from benign lesions. It has been stated that the disappearance of an ulcer under clinical management is not very helpful in differentiating between benign and malignant ulcers. We have used another sign, which is based more on theory than on proof; therefore, I cannot state it as a fact. We put the patient on clinical management. We watch the crater disappear. The crater of an ulcerating carcinoma in the presence of free acid will fill up with carcinomatous tissue and the crater disappear, when the acid is neutralized. The infiltration, however, remains. We can observe this infiltration both at roentgenologic and gastroscopic examination. In many benign ulcers the infiltration disappears. We now use this criterion. If the ulcer and the infiltration disappear during medical treatment we are probably dealing with a benign ulcer. The reverse is not necessarily true for inflammatory infiltration may remain after a benign ulcer heals.

In the past some gastroscopists have voiced their ability to determine the operability of carcinoma. New surgical techniques I think pretty well rule out any chance for the roentgenologist or the gastroscopist to determine the operability of a carcinoma. Distant metastases are apparently the only thing that prevent the surgeon from removing a neoplastic process. At our hospital the surgeons take out the pancreas, the spleen, the lower end of the esophagus; in fact, they sometimes clean out the whole left upper quadrant in removing a carcinoma of the stomach which has infiltrated the surrounding structures.

DR. RENSHAW (closing). It is a pleasure to have been invited as a gastroenterologist to appear on this program. I wish to express my gratitude for the privilege of being with you, and I wish to thank the discussants for their kind remarks.

The question of the personal factor in interpretation of a lesion has been presented. I tried to emphasize it in my paper, stating that our roentgenologic examinations were done by several examiners each with a different degree of experience and skill. A certain number of the indeterminate or unsatisfactory roentgenologic examinations were done by the less experienced examiners. The gastroscopic interpretations, on the other hand, were all done by one person.

The differentiation of an erosion from a small shallow ulcer is not always easy by gastroscopy. Our experience has been similar to Templeton's and Schindler's in that the majority of so-called gastric ulcers which were visualized by the gastroscopist and not by the roentgenologist appeared to be shallow. However, it should be emphasized that it is extremely difficult to judge the depth of an ulcer crater gastroscopically. I believe the gastroscopic appearance to be more shallow than is actually the case. I recall one very interesting and instructive case wherein we visualized an ulcer that was not seen roentgenologically. This patient had been twice examined elsewhere by a competent roentgenologist and at least twice by another gastroscopist. An ulcer was demonstrated by the gastroscopist but not by the roentgenologist. Several months later the patient presented himself at the Cleveland Clinic. Knowing the previous history, our roentgenologist made a special effort to demonstrate the lesion but was unsuccessful, yet I had no difficulty in visualizing an ulcer crater of moderate depth in the lesser curvature near the angularis. We had the same experience at two subsequent examinations. When the patient submitted to surgery and a partial gastric resection was done, we found an ulcer crater 2.5 by 2 cm. in diameter and 0.5 cm. deep with early malignant changes in the edges. One would think an ulcer of this size should have been visualized on at least one of the roentgenologic examinations. I have no satisfactory explanation for the roentgenologic failures.

Small erosions which might be confused with, and which should be differentiated from, mucus patches or barium flecks would not be confused with or classified as shallow gastric ulcers.

Dr. Templeton has asked about the clinical symptoms of gastritis in 25.6 per cent of our group where we thought gastroscopy had made a primary contribution. I can answer that by saying that I went over a series of 300 cases which are included as the first 300 of this present series of 842 patients. I excluded cases of cholecystic disease and other conditions which might account for the patient's symptoms. Even patients who used cathartics in the presence of irritable colon or patients with a large functional element were excluded. In the remaining group where were not over 6 per cent in whom we thought the gastritis was of clinical significance. I have repeatedly emphasized to my colleagues that the mere finding of mucosal changes does not mean active disease. The gastric mucosal changes should be regarded as roentgenologists regard a duodenal bulb deformity. Bulb deformity without a visible crater may mean an old ulcer or it may mean an active duodenal ulcer. Gastric mucosal changes may mean active or inactive disease. It is up to the clinician to decide whether the changes represent active disease or not.

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COMMON CONGENITAL ANOMALIES OF THE BONY THORAX

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ALTHOUGH several articles describing congenital anomalies of the bony thorax have appeared in the foreign literature, search has failed to reveal any survey of the subject in the American literature*, and only one paper on this theme in the English literature.[1] It seemed of interest, therefore, to investigate the nature and incidence of congenital variations in a series of routine chest roentgenograms.

Material. The roentgenograms studied were taken as part of the entrance physical examination given yearly to the freshman class at Dartmouth College. The subjects are males, almost all of the age of nineteen or twenty years. Since White, in 5,000 routine hospital chest roentgenograms found an incidence of 1 per cent in both males and females, the figure for percentage incidence found in this series would presumably be applicable to both sexes. For individual anomalies, however, the same author found variations in incidence between males and females. Two thousand roentgenograms were reviewed to provide the material studied.

Etiology. In the development of the embryo, the primitive mesoderm undergoes a process of segmentation into a series of somites. A portion of the mesoderm of each somite differentiates into the sclerotome, from which the vertebra is developed. The rib is derived from an outgrowth of the developing vertebra. It is generally held that anomalies occur as a result of irregular segmentation in the first step of division of the mesoderm into somites.

Incidence. A total of 56 anomalies was observed, an incidence of 2.8 per cent. The type and distribution of the anomalies is given in Table I.

TABLE I
TYPE AND NUMBER OF ANOMALIES

Bifurcation	20
Cervical rib	10
Rudimentary first rib	10
Flaring	6
Fusion	6
Bridging	4
	56

Bifurcation. Bifurcation of the anterior end of a rib occurred in 20 cases, all involving the third, fourth, or fifth rib and almost equally distributed among the three (Table II). The majority (13) were on the right

TABLE II
BIFURCATION

Third rib	6	Right 13
Fourth rib	8	Left 7
Fifth rib	7	

side. The notch formed by the bifurcation may be shallow and asymmetrical as in Figure 1, or deep and symmetrical as in Figure 2. One case (Fig. 3) showed bifurcation of two ribs.

Flaring. Flaring of the anterior end of the rib was found in 6 cases, distributed as shown in Table III. A typical example is seen in Figure 4. The slight notching in 1 case (Fig. 5) suggests a close relationship between this and the preceding anomaly.

Fusion. Fusion of two adjacent ribs occurred in 6 cases, all but one in the first two ribs, and none below the third (Table IV). Fusion in all cases was incomplete,

* Since the preparation of this paper, the following papers have been published: Steiner, H. A. Roentgenologic manifestations and clinical symptoms of rib abnormalities. *Radiology* 1943, *40*, 175; Etter, L. E. Osseous abnormalities of the thoracic cage seen in forty thousand consecutive chest photoroentgenograms, AM. J. ROENTGENOL. & RAD. THERAPY, March, 1944, *51*, 359.

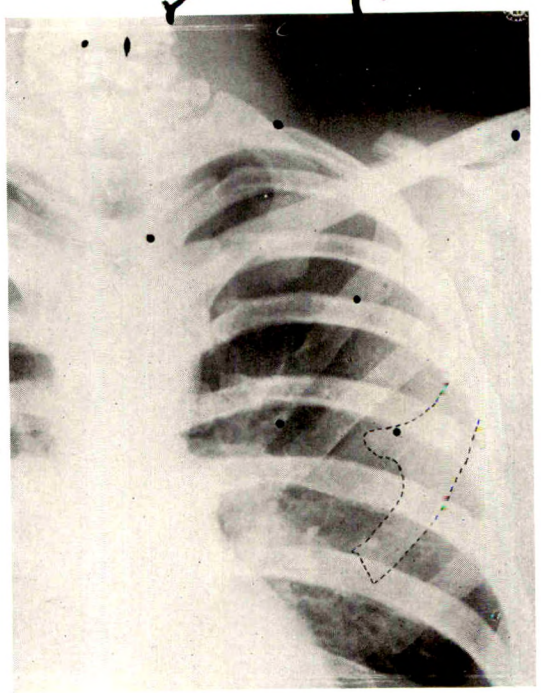


FIG. 1. Bifurcation.

with a bifurcated anterior extremity (Fig. 6). Two cases presented multiple anomalies: one, fusion of the first and second ribs, incomplete development of the fourth, and flaring of the fifth (Fig. 7); the other, fusion of the first and second ribs, associated with a hemivertebra (Fig. 8).

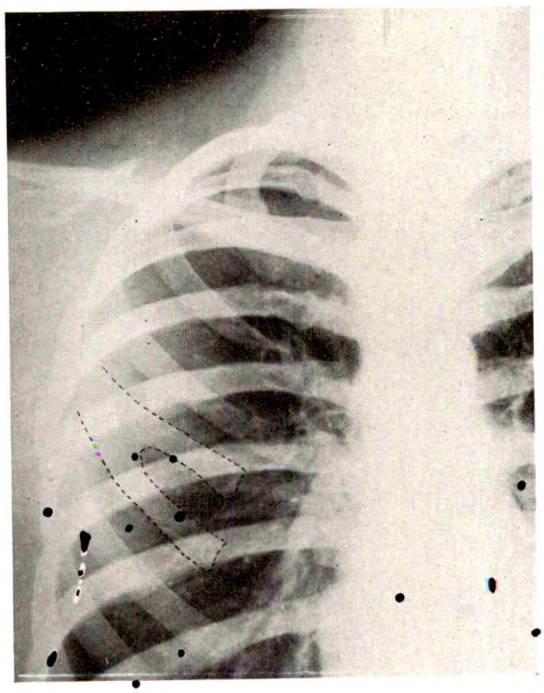


FIG. 2. Bifurcation.

TABLE III

FLARING

First rib	1	Right	4
Fourth rib	2	Left	2
Fifth rib	3		

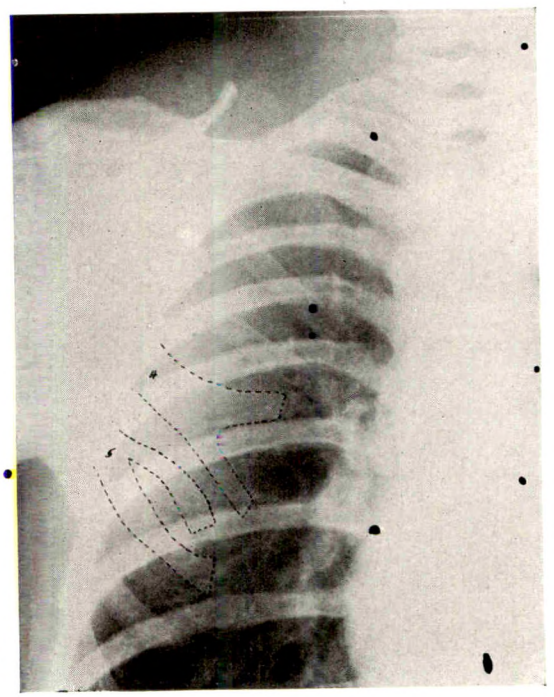


FIG. 3. Bifurcation of two ribs.

Bridging. Apparently closely allied to the preceding, bridging between two ribs may be complete as in Figure 9 or incomplete as in Figures 10 and 11. Four cases were observed, distributed as in Table V.

TABLE IV

FUSION

First and second ribs	5	Right	4
Second and third ribs	1	Left	2

TABLE V

BRIDGING

First and second ribs	3	Right	2
Second and third ribs	1	Left	2

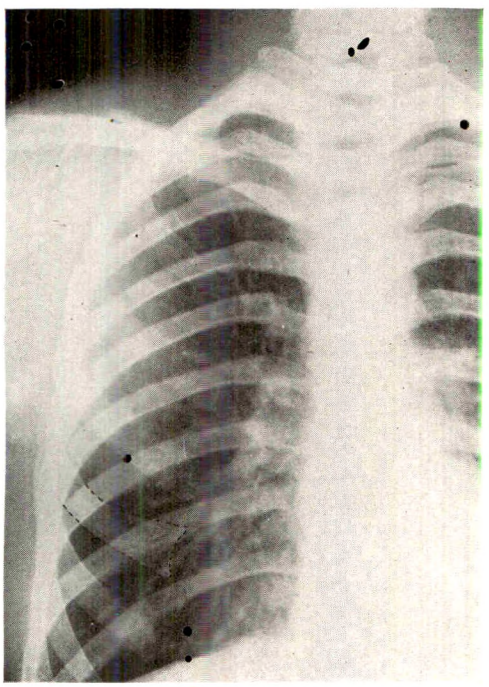


Fig. 4. Flaring.

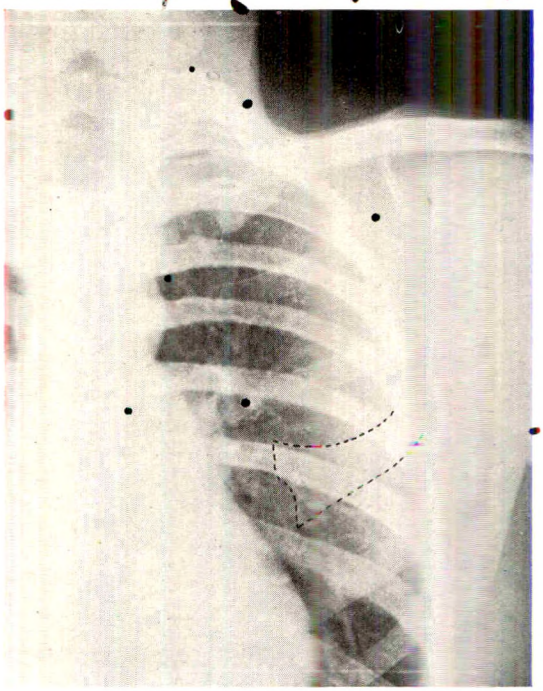


Fig. 5. Flaring.

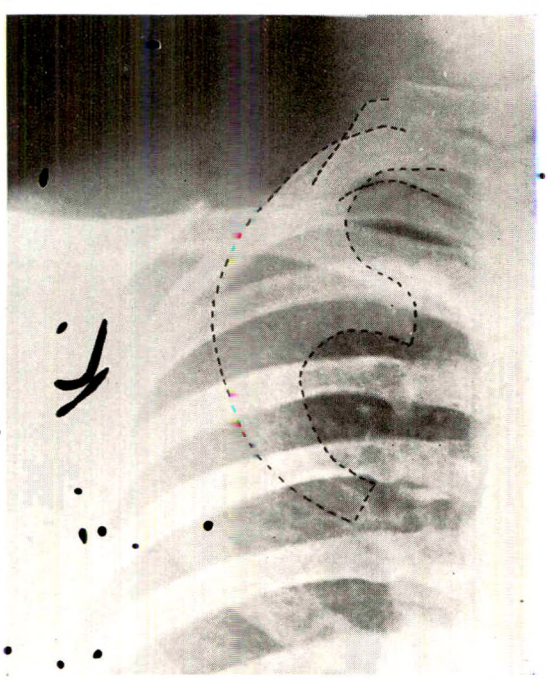


Fig. 6. Fusion.

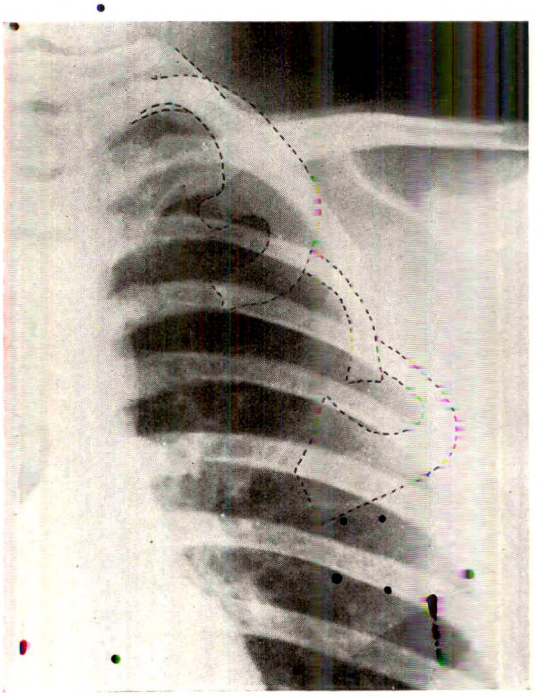


Fig. 7. Multiple anomalies.

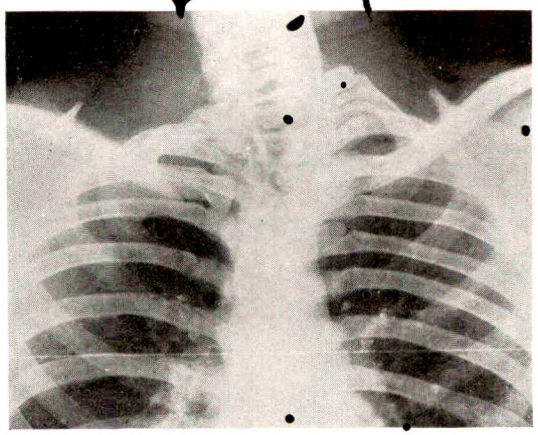


FIG. 8. Fusion associated with hemivertebra.

Cervical Rib and Rudimentary First Rib. Ten cases of each of these variations occurred, the differentiation between the two being made by observing the relationship of the anterior end of the rib to the sternum. It will be seen by reference to Tables VI and VII that the majority of cases of cervical rib were bilateral, whereas the majority of rudimentary first ribs were unilateral. In the case of cervical ribs, the majority united with the first rib; with rudimentary ribs, the anterior end was free in the majority of cases.

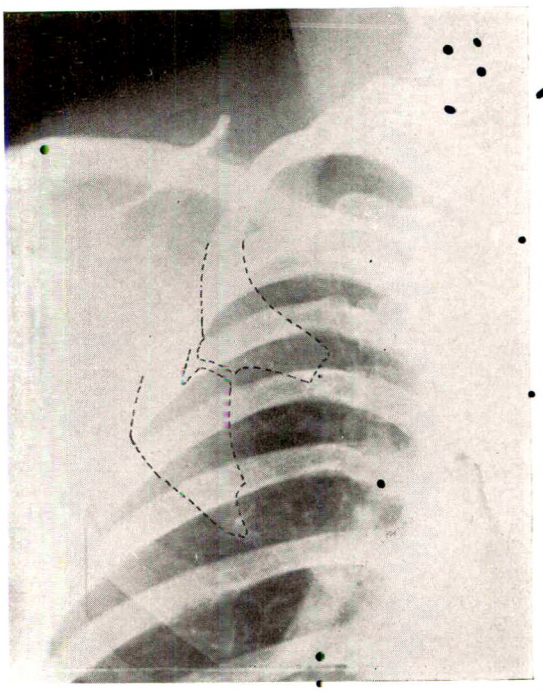


FIG. 10. Partial bridging.

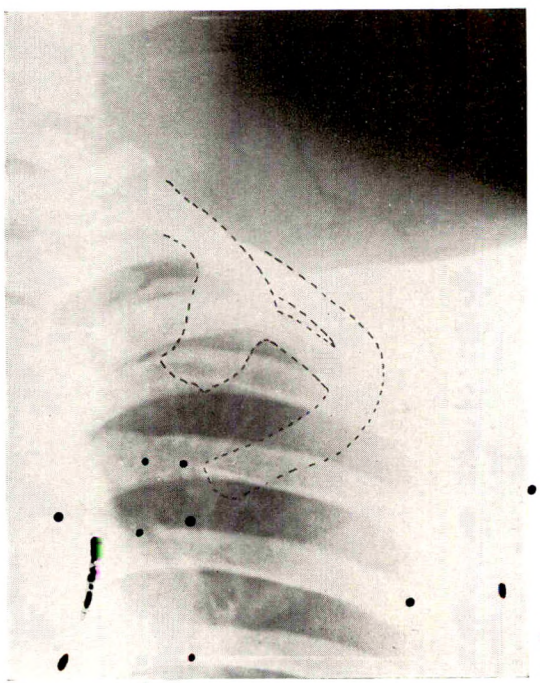


FIG. 9. Bridging.

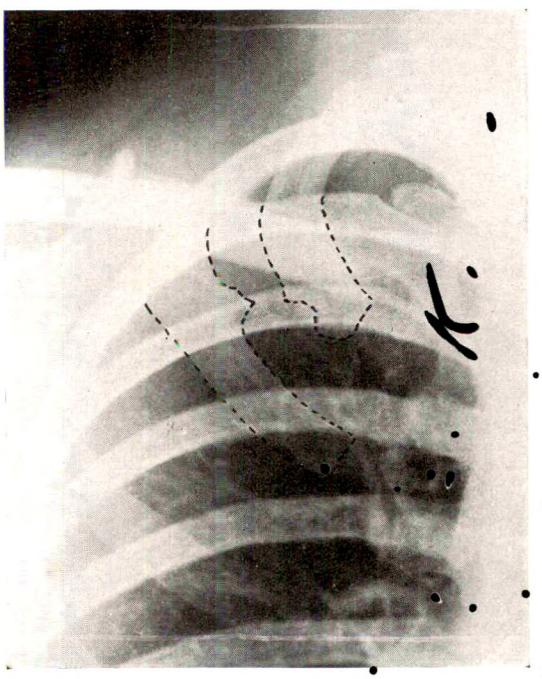


FIG. 11. Partial bridging.

TABLE VI

CERVICAL RIB

Bilateral	7	Free end	5
Right	3	Union with first	11
Left	0	Articulation with first	1

TABLE VII

RUDIMENTARY FIRST RIB

Bilateral	4	Free end	11
Right	2	Union with first	2
Left	4	Articulation with first	1

"Fracture" of the First Rib. One cervical rib and one first rib revealed a solution of continuity as shown in Figures 12 and 13.

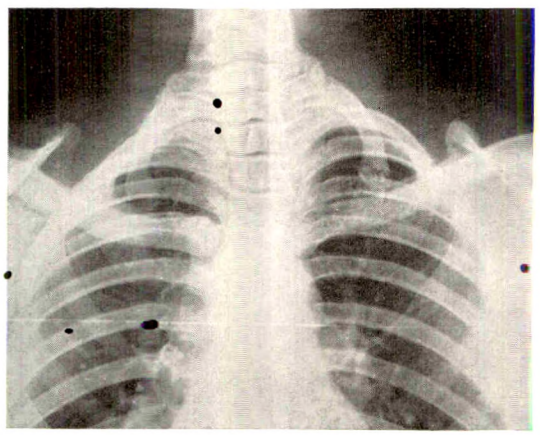


FIG. 12. Anomaly of first rib simulating fracture.

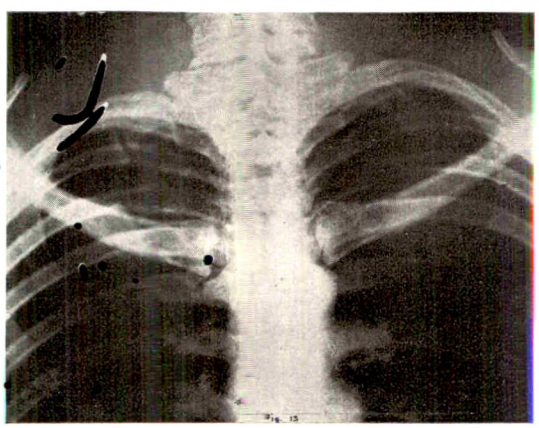


FIG. 13. Anomaly of first rib simulating fracture.

This is similar in appearance to some of the lesions which have been described in several papers as fracture of the first rib. It has

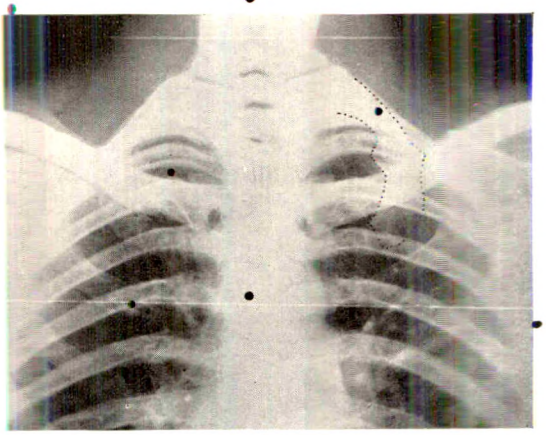


FIG. 14. Possible anomaly simulating healed fracture.

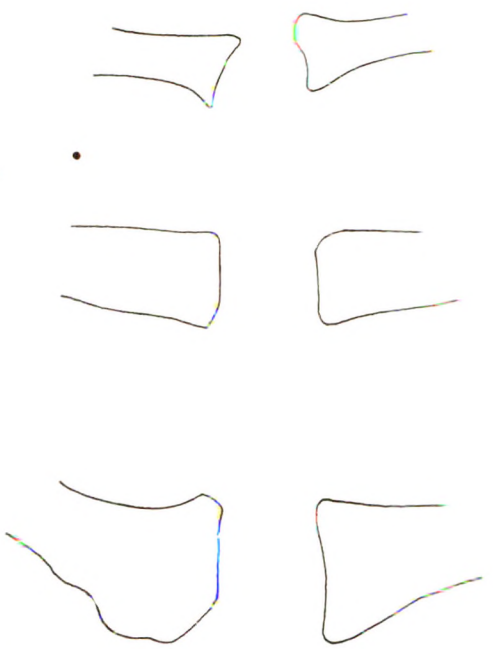


FIG. 15. Variations in medial end of clavicle.

been considered that such a fracture can be produced by muscle traction as well as by direct or indirect trauma. Figure 14 shows an appearance which is apparently the end-result of the same process. In none of the 3 cases was a history of trauma or disability obtained. It is recognized that this by no

means rules out the possibility of fracture, since the symptoms may not have been severe enough to fix the incident in the patient's memory. Certain factors in addition to the negative history, however, raise the question as to whether we may be dealing with an anomaly of some type. The line of diminished density is broader and more irregular than is usually seen in fracture of other ribs. Non-union of rib fractures is rare, and 1 case (Fig. 13) showed the same findings at two different examinations one year apart. We have seen that the cervicothoracic junction is a frequent site of congenital anomaly, and in 1 case there was an associated fusion of the fifth and sixth cervical bodies. These conditions, therefore, suggest the possibility that some "fractures" of the first rib may actually be cases of congenital anomaly to which attention is called by a superimposed injury.

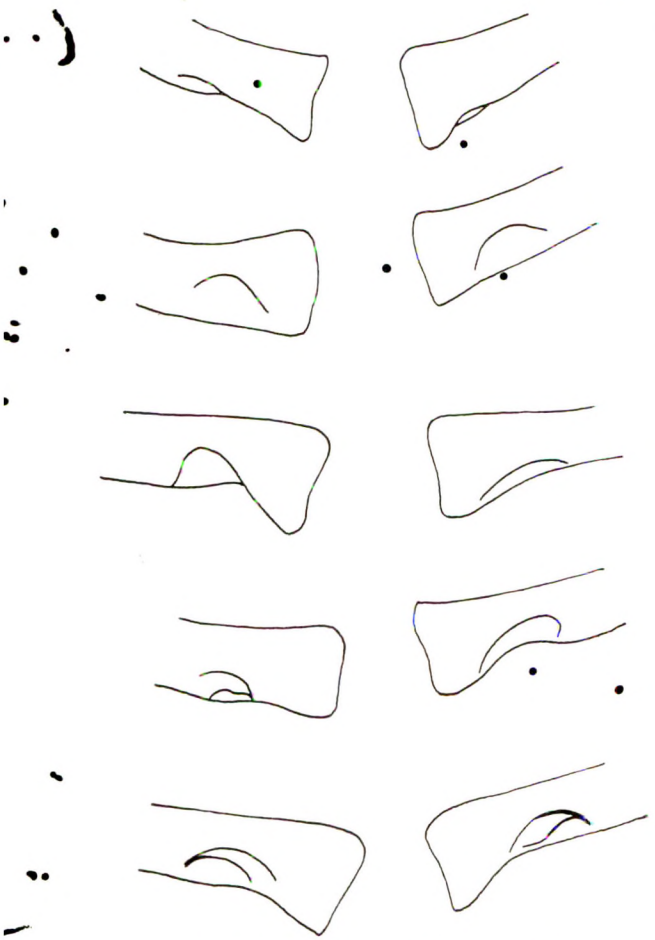


Fig. 16. Rhomboid fossa of clavicle—bilateral.

TABLE VIII

RHOMBOID FOSSA OF CLAVICLE

Total number in 500 subjects	121	24%
Bilateral	71	58%
Right	26	21%
Left	24	21%

Rhomboid Fossa of the Clavicle. Although this entity does not strictly fall under the heading of congenital anomaly, the recent publications of Pendergrass and Hodes[2] and of Shulman[3] stimulated a study of its occurrence. The shape and size of the medial end of the clavicle varied markedly as indicated

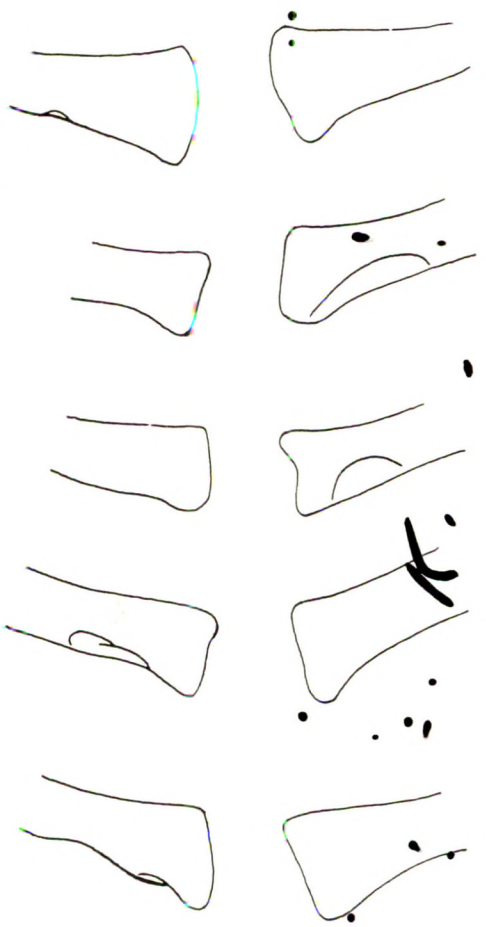


Fig. 17. Rhomboid fossa of clavicle—unilateral.

in Figure 15. The fossa was considered to be present when a definite double contour could be seen on the roentgenogram. A study of 500 consecutive cases revealed an incidence of 24 per cent (Table VIII). Of these, 58 per cent were bilateral and 42 per cent unilateral, equally divided between right and left. The fossa can be shallow or deep, and may be situated at any point from 1.0 cm. to 3.0 cm. from the sternal end of the clavicle. Illustrations of the various types are shown by the tracings reproduced in Figures 16 and 17.

Calcification of the First Costal Cartilage. Another coincidental survey was made to investigate the incidence of calcification of the first costal cartilage. Islands of calcium deposit were a frequent finding, but the early stage of progressive calcification, seen usually as an irregular line along the lower border of the cartilage, was uncommon, occurring in 4 per cent of 500 consecutive roentgenograms. A series of 100 roentgenograms of medical students averaging three to four years older, on the other hand, showed calcification in a greater or lesser degree in 42 per cent. Only one case showed calcification in the other costal cartilages, and in this one the first cartilage was almost completely calcified. From these figures it appears that the beginning of calcification of the first costal cartilage occurs in males in the earliest years of the third decade, and precedes calcification of the other cartilages.

SUMMARY

Congenital anomalies of the bony thorax occurred in 2.8 per cent of two thousand male college students. These anomalies are tabulated and illustrated.

Rhomboid fossa of the clavicle was present in 24 per cent. Its various forms are illustrated.

Calcification of the first costal cartilage was found in 4 per cent of college freshmen, but in 42 per cent of first and second year medical students.

Evidence is presented which suggests that the appearance described by various authors as fracture of the first rib may in some cases be due to a congenital anomaly.

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2. PENDERGRASS, E. P., and HODES, P. J. Rhomboid fossa of clavicle. AM. J. ROENTGENOL. & RAD. THERAPY, 1937, *38*, 152–155.
3. SHULMAN, S. Rhomboid depression of the clavicle. *Radiology*, 1941, *37*, 489–490.

IMPORTANCE OF THE ROENTGEN EXAMINATION IN THE DIAGNOSIS OF ADENOMA OF THE PROSTATE*†

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IN THE masculine preclimacteric and climacteric (fourth to fifth decades) the prostatic part of the urethra is affected by a tumor formation which grows progressively. Formerly called prostatic struma (Tsunodas), suburethral tumor (Marquis), periurethral adenoma (Motz), prostatic adenoma (Legueu), hypertrophy of the prostate (Mercier), periprostatic adenoma (Rubritius), it is now more properly called adenoma of the prostatic urethra.

The substrate of this structure is an intense cellular hyperplasia of the submucous or suburethral glands (of endodermal origin), sometimes called Lendorf's accessory periurethral glands.

These glands are distributed in groups from the internal ring to the submontanal part of the prostatic urethra (Fig. 1 and 2).

Horn Orator has made a systematic classification of these groups as follows: (*a*) the trigone or subcervical group (Albarran's group); (*b*) colliculus group (lateral group); (*c*) distal group (ventral group or group of the head of the prostatic urethra).

The clinical forms of adenoma of the prostate are characterized by the solitary or combined growth of these cell groups (Fig. 3): (*a*) total adenoma (originating from the lateral, distal and subcervical groups); (*b*) middle lobe or Home's lobe (from the subcervical or trigone group); (*c*) micro-adenoma (miniature form) (from the subcervical group with intrasphincteric growth); (*d*) solitary distal lobe (distal group, or that of the head of the prostatic urethra).

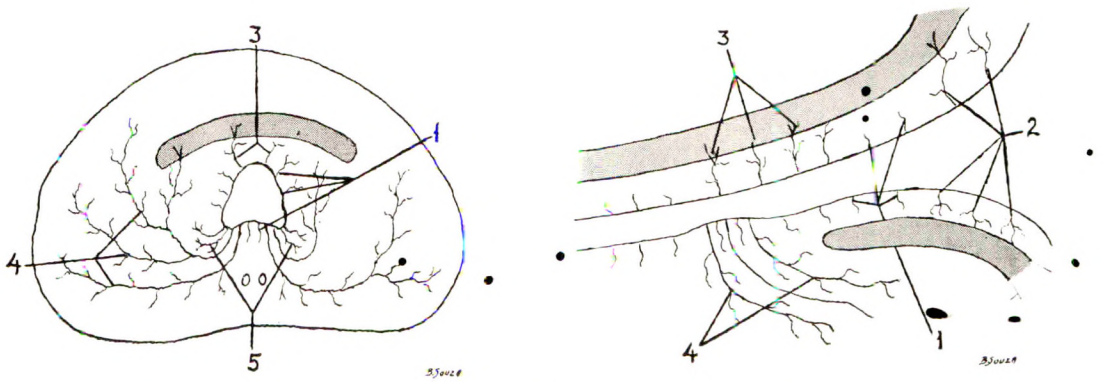


FIG. 1 and 2 (according to Jacoby). *1*, urethral mucous glands; *2*, submucous glands (trigone or subcervical groups); *3*, submucous glands (ventral or distal group); *4*, prostatic glands, properly so-called; *5*, submucous glands (colliculus group).

On the other hand, the prostatic glands properly so called (of mesodermal origin) when affected by tumoral new-growth (adenoma) may cause enlargement of the organ with the same clinical manifestations as total adenoma of the glands of Lendorf (submucous glands).

Here two varieties may be differentiated,

* Presented before the April 15, 1942, session of the Medical and Surgical Society of Sao Paulo, Brazil, and the Section of Urology of the Paulist Association of Medicine, session of April 25, 1942.

† Translation made by Dr. Audrey G. Morgan, Fort Scott, Kansas.

from both the clinical and anatomo-pathological points of view: (*a*) "very minute intraprostatic" adenoma; (*b*) fibroadenoma of the prostatic glands (Albarran).

In the growth of the tumor, whether of the variety originating from the submucous glands (total) or of that originating from the prostatic glands properly speaking, the direction of growth may be vesical, subvesical or perineal (Fig. 4).

This difference is of great interest, particularly with reference to indications for operative treatment, since within certain limits it indicates the route to be followed: (*a*) suprapubic transvesical (high section) (Freyer, Fuller, Harris); (*b*) perineal (transverse, median, lateral perineotomies) (Zuckerkandl, Albarran, Young, Wildbolz, Wilms, Berndt, Lowsley); (*c*) ischiorectal (Voelcker); (*d*) transurethral (endoscopic resection).

With reference to the cause of this intense hyperplasia, whether of the suburethral or submucous glands, or of the prostatic glands properly speaking, different theories were formerly advanced (arteriosclerotic, inflammatory, neoplastic, degenerative) but at present the development of adenoma of the prostatic glands, properly speaking, is thought to have the same origin as that of tumors in general, while adenoma of the suburethral or submucous glands is thought to be due to a hormonal disequilibrium due to change of life (hormonal theory).

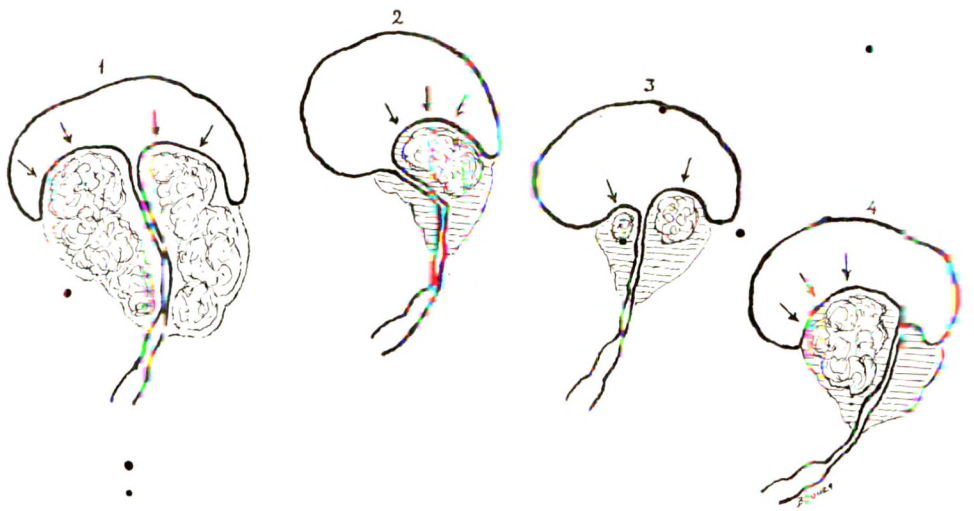


Fig. 3. Clinical forms of adenoma of the prostate. *1*, total adenoma; *2*, middle lobe or Home's lobe; *3*, microadenoma (miniature form); *4*, distal lobe.

I refer on this point to the ideas of Lower and his collaborators at the Cleveland Clinic, those of Laquer of Amsterdam and those of Deming of Yale University.

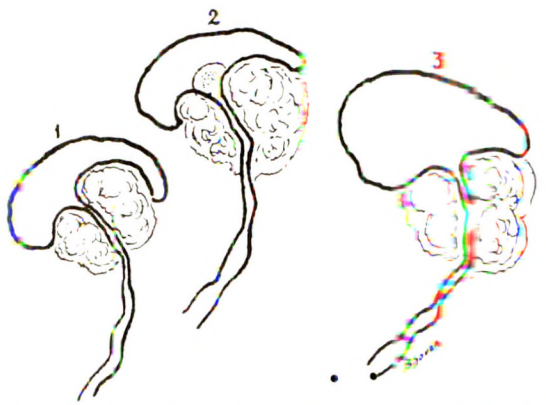


Fig. 4. Forms of growth of adenoma of the prostate. *1*, intravesical adenoma; *2*, part intravesical part subvesical; *3*, subvesical.

Lower and his collaborators demonstrated an interrelationship between the anterior lobe of the hypophysis, the testicles and the secondary sexual organs of certain

animals; and they assume that this correlation must exist also in man.

McCullagh, Walsh, Moore, Owen, Cutler, Lower and de Jongh in well conducted experiments found that the testicle produces two hormones: the first is androtin, a male sexual hormone, produced by the interstitial cells, which controls the development and maintenance of the secondary sexual glands. Its production is regulated by the anterior lobe of the hypophysis. Hyperfunction of the anterior lobe of the hypophysis stimulates the testicle in such a way that excessive production of androtin causes hyperplasia of the submucous glands, or so-called adenoma of the prostate.

The second hormone, inhibin or contruin, produced by the germinal cells, controls the hypophysis. Normally the germinal cells secrete enough inhibin to prevent excessive secretion of the hormone of the anterior lobe of the hypophysis, but as soon as functional imbalance takes place, with decreased function of the germinal cells, which appears to be caused by change of life (preclimacteric and climacteric) the decreased amount of inhibin permits of greater activity of the anterior lobe of the hypophysis which stimulates excessive production of androtin by the interstitial cells which is responsible for the glandular hyperplasia of adenoma of the prostatic urethra.

According to Lower and his collaborators therefore the absence or decrease of inhibin permits of hyperactivity of the anterior lobe of the hypophysis which stimulates excessive production of androtin, which is responsible for glandular hyperplasia of the suburethral glands or Lendorf's accessory glands.

Laquer of Amsterdam, however, believes that the genesis of adenoma is quite different from this; there is a factor of hormonal disequilibrium, but it is between the androgenous and estrogenic hormones.

In the age of prostatism (the preclimacteric and climacteric) the two sexual hormones are in a condition of balance with each other, with the hormone of the anterior hypophysis and with the hormone of the suprarenals. The hyperplasia of the submucous glands, the so-called benign hypertrophy of the prostate, according to him would be caused by deficiency of the masculine hormone (which would inhibit the hyperplastic action of the estrogenic hormone) and of other gonadotropic substances not inhibited by it. There would therefore be a predominance of estrogenic and gonadotropic hormones.

In addition to these two theories there is that of Deming that the hyperplasia of adenoma of the prostate is caused by the invasion of solid fibromuscular masses from the muscular wall of the urethra into the gland ducts. These solid fibromuscular masses, originating from the müllerian ducts are said to acquire this invasive growth from stimulation by estrogenic hormones. Here, too, there would be disequilibrium between the androgenic and estrogenic hormones.

As soon as the growth of adenoma of the prostatic urethra has begun the patient begins to feel mild symptoms which are either disregarded or attributed to incidents in the daily life, such as food, occupational activity or excessive drinking or sexual activity. In this premonitory stage or stage of mild irritation, prostatic congestion predominates with nocturnal urinary disturbances (dysuria and pollakiuria).

In a more advanced stage the local symptoms begin (difficulty and pain on micturition) which grow worse from day to day with repeated attacks of hypertonia of the sphincter, impossibility of emptying all the contents of the bladder (residual urine), polyuria, particularly at night, and symptoms of general exhaustion. The disturbances of bladder dynamics progress (hypertonia, hypotonia, even atony) to such a degree that frequently the patients have an attack of acute retention (functional impossibility of emptying the bladder) which is certain to be followed by infection. The aggravation of these symptoms results before long in inability to empty

the bladder spontaneously. Its musculature loses its capacity for contraction, becomes excessively distended, and complete retention begins. In some cases even in this period there is a slight contractile reaction on the part of the bladder and the urine regurgitates through the urethra, causing paradoxical ischuria. The excretory tracts and the kidneys having become involved, it is not long before insufficiency of the kidneys results, there are repeated attacks of pyelonephritis, and uremia and urosepsis complete the final picture of all these complications caused by adenoma of the prostatic urethra.

The patient may come to the general physician or the specialist in any of these stages of the disease. It is best to examine him in the early stage of the disease in order to make an accurate diagnosis both with regard to the clinical manifestations of tumor of the prostate and of the disease itself with its local and general disturbances and kidney complications. From this examination the therapeutic indications will be established, which may eventuate in surgical operation, either palliative, conservative or radical.

Our routine examination for a long time has been as follows:

(*a*) History of the symptoms which have aroused suspicion, together with anything indicating disturbances of the neck (the so-called cystectasias) or massive lithiasis of the prostate, or prostatitis.

(*b*) Digital exploration by the methods of Picker, Sellei, Fuller and Voelcker, Spencer, and others, which shows the size and consistency of the prostate, but which does not enable a diagnosis to be made of the middle lobe, of micro-adenoma in an atrophied prostate of the ventral lobe, or even of differentiation between prostatitis, massive lithiasis of the prostate and adenoma in certain stages of its development.

(*c*) Exploration of the urethra (with a sound) which shows the length of the prostatic urethra, makes it possible for the examiner to feel the obstruction caused by a tumor of considerable size and supplies residual urine and material for the investigation of certain complications such as urinary infection, but is not sufficient for a complete examination.

(*d*) Urethrocystoscopy (instruments of McCarthy, Lowsley and Peterson, v. Lichtenberg, and others), which is dependent on permeability of the urethra and which gives a clear view of the protrusion of lobes of an adenoma into the enlarged and deformed prostatic urethra, of the middle and ventral lobes and of other complications and modifications resulting from the adenoma itself (pulsion diverticula, true diverticula, hypertrophy of the inter-ureteric torus, trabeculated bladder, inflammatory conditions, changes in the ureteral orifices, lithiasis of the bladder, etc.). But this method of examination does not give a clear picture of the pelvic localization of the tumor, it does not permit of differentiation between fibrosis of the neck and beginning hypertrophy of the middle lobe and it causes trauma, and hemorrhage. It increases the suffering of the patient in advanced stages and is impractical in large tumors in which it is accompanied by hemorrhage in spite of continuous irrigation, which makes false routes between the lobes of an adenoma developing in the membranous urethra, and in prostatics and patients with urinary infection frequently causes attacks of ascending pyelonephritis (trauma, reflux).

(*e*) Roentgen examination which until recently was restricted to roentgenography of the prostate with an opaque sound, to Burckhardt-Floerckern's aerocystography and to cystography with oily solutions or suspensions as a contrast medium which were sometimes irritating, but recently has included intravenous urography for the diagnosis of dynamic disturbances brought about by adenoma of the upper excretory passages and of the functional condition of the kidneys* (Fig. 5 and 6). Retrograde cystography almost always shows incidents in the development of the adenoma, such

*Sometimes the cystogram shows the size of a prostatic tumor which has extended into the bladder.

as pulsion diverticula, true diverticula and finally residual urine and the appearance of the paretic bladder.

Roentgenography is not concerned with demonstrating the different clinical forms of adenoma, as such determinations can be

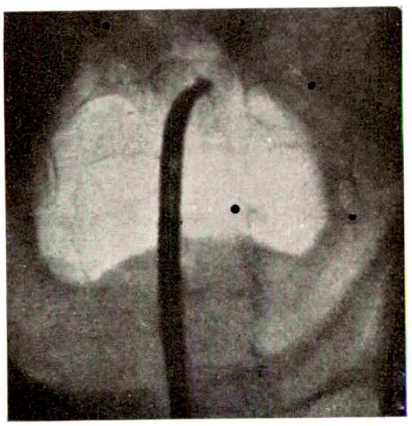


FIG. 5. Roentgenogram of the prostate with opaque sound. (According to Casper and Picard.)

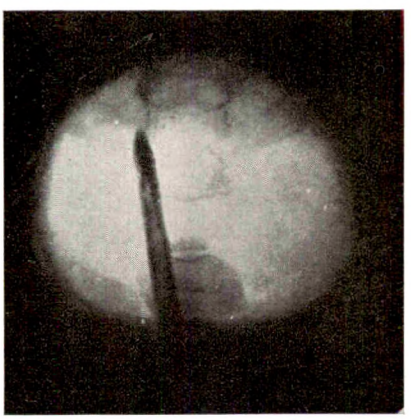


FIG. 6. Aero-cystography in adenoma of the prostate. (According to Casper and Picard.)

made by cystography (with air, retrograde or intravenous urography). This accomplishment is due solely to retrograde urethrocystography, which shows the undeniable importance of roentgen examination of patients with adenoma of the prostate.[8]

Technique of Urethrocystrography.

The technique of urethrocystography according to the method described in a "preliminary note" before the Urological Section of the Paulist Medical Association, August 25, 1940, consists essentially of:

(*a*) Previous filling of the bladder for the purpose of showing its entire outline, particularly that of the basal portion; 60 to 80 cc. of the contrast medium is injected. The amount varies in the individual case of adenoma of the prostate, depending on the size of the tumor when previous routine examinations (palpation, exploration of the urethra, urethrocystoscopy) have already shown its size. When they have not, the first roentgenogram will indicate whether

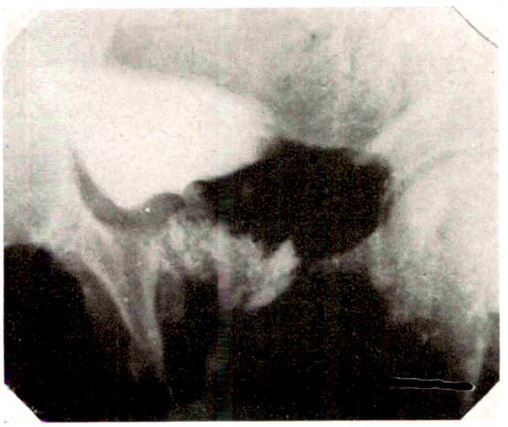


FIG. 7. Normal image of the neck. Dilated prostatic glands, prostatic urethra not elongated (profile roentgenogram).

it is necessary for the amount to be increased (in large adenomas growing into the bladder, for example). When, on the other hand, there is a large amount of residual urine, the bladder should be emptied before the contrast medium is injected.

(*b*) The filling of the whole of the urethra (15 to 20 cc. of contrast medium). For this purpose a syringe of 20 cc. capacity is used which is adapted for the purpose by using a rubber tip which occludes the opening of the urethra.

(*c*) A roentgenogram made exactly during the terminal part (10 cc.) of the urethral injection.*

* Based on the principle of cerebral arteriography of Egas Moniz

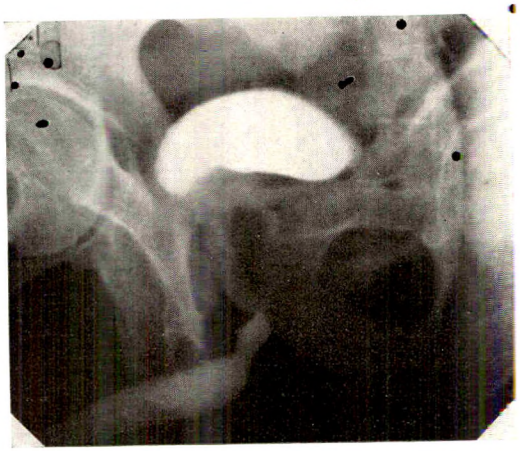


FIG. 8

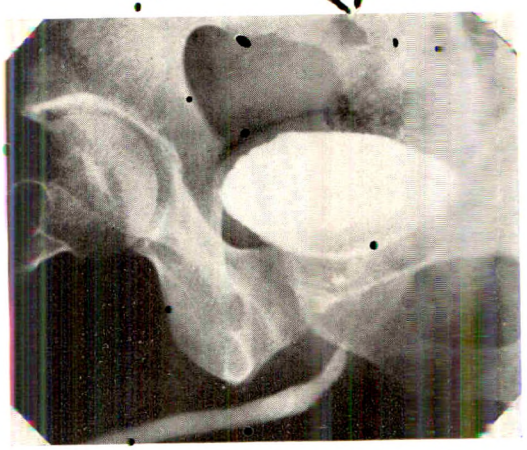


FIG. 9

FIG. 8. Urethrocystography in chronic prostatitis. Elevation of the base of the bladder by increase in the size of the prostate. Prostatic urethra without the changes of adenoma (profile roentgenogram).

FIG. 9. Urethrocystography in chronic prostatitis. Elevation of the base of the bladder by increased size of the prostate. Prostate glands slightly dilated. Prostatic urethra not elongated (profile roentgenogram).

No force should be used. The injection should not be made too rapidly or too slowly. Otherwise the roentgenogram will not show a clear picture of the prostatic urethra nor a perfect image of the "internal sphincter."

Positions. For the purpose of systematizing the examination two positions are used: (*a*) lateral oblique, or semiprofile, and (*b*) anteroposterior or frontal. In the former the patient is placed in dorsolateral decubitus, preferably right, with the thigh flexed and in forced abduction with relation to the opposite leg which is extended. In the anteroposterior or frontal position the patient lies in dorsal decubitus. With the roentgen tube centered on the pubis the roentgenogram is made according to the requirements peculiar to each particular form of apparatus (milliamperage, exposure time, kilovoltage, which vary according to the thickness of the patient's body).

Contrast Media. There has been a great deal of discussion of the qualities of the various contrast media used in the examinations and of which are to be preferred (thorotrast, uroselectan, and perabrodil, lipiodol, 20 per cent neo-iodipin, 25 per cent sodium bromide, 35 per cent sodium iodide, suspension of rec barite, barium sulfate and 30 per cent citobarium); some of them dangerous, others irritating. We prefer a suspension of luxobarium,* prepared by the Spic Laboratory of Sao Paulo, because of its cheapness, ease of preparation, chemical harmlessness, opacity, and so forth. The suspension is prepared especially for us in the proportion of 60 gm. powder to 100 cc. boiling water.

Roentgen Diagnosis. When the two roentgenograms, profile and frontal, are obtained the roentgen diagnosis is made† (Fig. 7, 8 and 9). It is the urologist who is best fitted to make this examination and give the most accurate interpretation. He knows the history of the patient and has already certain preliminary examinations and knows the object of the examination and what is to be disproved or confirmed.

The points to be considered are: (*a*) the characteristics of the bulbar filling; (*b*) the form and relations of the membranous urethra to the adenoma; (*c*) the elongation, deformities, dilatations and angulations of the prostatic urethra (supra- and infra-montanal); (*d*) the arrangement of the "internal sphincter" in relation to the bladder

* The base is barium sulfate.

† We refer here to the roentgen diagnosis of urethrocystography. It is well to remember that in cases in which cystoscopy is impossible an orienting roentgenogram precedes urethrocystography for the verification of lithiasis of the bladder.

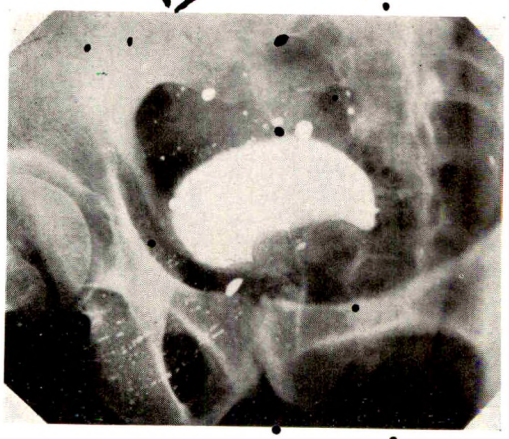


FIG. 10. Miniature form of adenoma of the prostate (profile roentgenogram). Two small nodules developed intravesically. Atrophy of the prostate gland (seventy-four years).

image of the urethra is short, or not elongated, since this form of adenoma occurs in a prostate of normal size or even more frequently in a prostate atrophied by senile involution. Sometimes the tumor projects into the base of the bladder and into the supramontanal part of the prostatic urethra, and sometimes the small tumor nodule develops into the opening of the

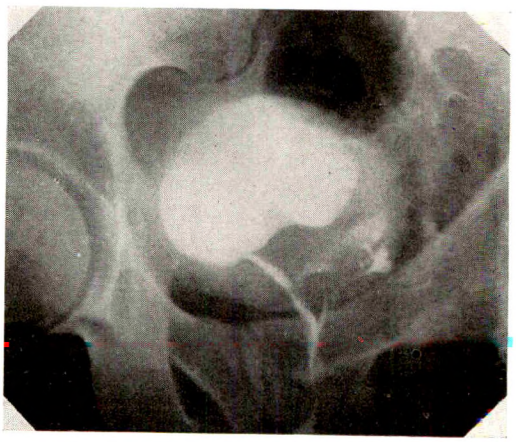


FIG. 11

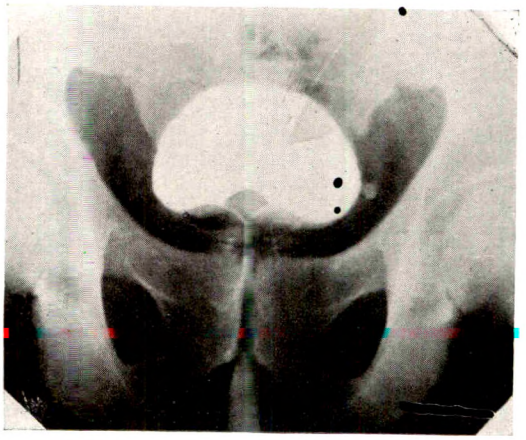


FIG. 12

FIG. 11. Outline of middle lobe (profile roentgenogram). Angulation of the prostatic urethra. Endoscopic appearance of fibrosis of the neck.

FIG. 12. Outline of middle lobe (frontal roentgenogram). Prostatic urethra not elongated. The lobe protruding between elevations of the prostate enlarged from prostatitis.

cavity; (*e*) the configuration of the elevations on the basal plane of the bladder; (*f*) the perfection, the clearness and the deformities of the outline of the bladder.

The roentgen diagnosis will be based on a minute observation of the above points.

It should be noted, however, that the characteristics of each segment examined vary with the stage of development of the prostatic tumor (adenoma) and its clinical form.

In the miniature form of adenoma the

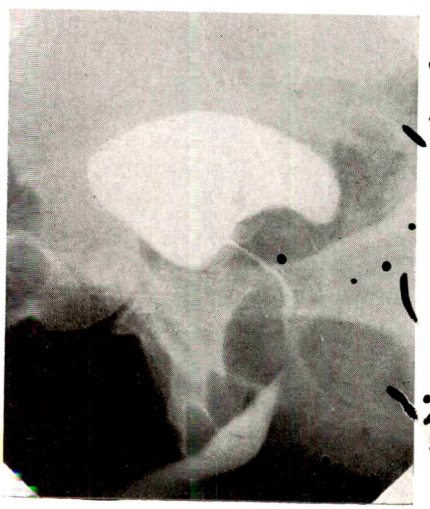


FIG. 13. Solitary middle lobe (profile roentgenogram). Angulation of the prostatic urethra. The "internal sphincter" is directed forward, the nodule protruding into the bladder.

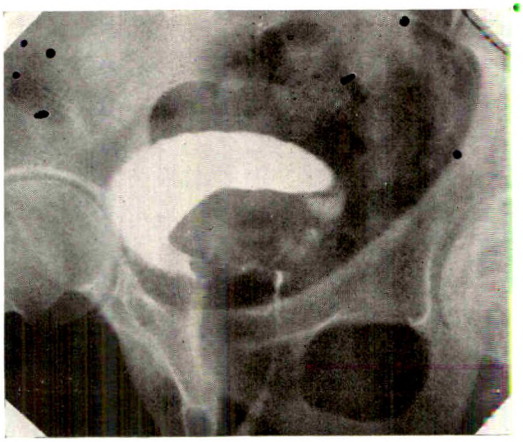


Fig. 14

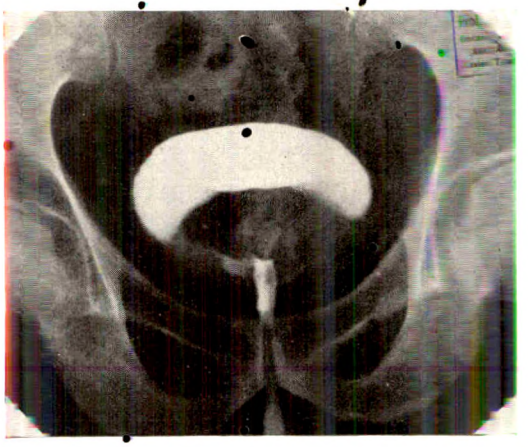


Fig. 15

Fig. 14. Middle lobe grown to large size (profile roentgenogram). Notable angulation of the prostatic urethra.
Fig. 15. The same case as Figure 14 (frontal roentgenogram). The image of the prostatic urethra is interrupted at the point of angulation.

sphincter of the bladder neck (Fig. 10). In these cases roentgen examination is of great aid in the interpretation of the urethrocystoscopic examination.

Three patients with this form of adenoma of the prostate were examined. In 2 the nodule, localized on the right side, was shown in the frontal roentgenogram as a protuberance into the supramontanal part of the urethra. In 1 case it protruded into the base of the bladder in the profile roentgenogram.

In solitary middle lobe in the profile roentgenogram it is always to be noted that the "internal sphincter" is directed forward, the nodule protruding, in a characteristic manner, back of the "sphincter" into the base of the bladder and varying greatly in size (depending on the stage of development) (Fig. 11, 12 and 13).

The image of the urethra, not elongated, protrudes into the supramontanal portion, making an angulation which is larger, the greater the development of the adenomatous nodule (Fig. 14). If there are accompanying lateral lobules with a subvesical or even intravesical growth, there is a varying degree of elongation of the prostatic urethra, and it may even be deformed and enlarged in diameter.

In the anteroposterior roentgenogram the nodule can be seen protruding into the base of the bladder between the borders of the "internal sphincter." The image of the urethra, elongated, broadened and with or without deformity, often shows an interruption at the point of angulation (Fig. 15).

We diagnosed the existence of a middle lobe in 10 patients; in 5 it was solitary and in 5 associated with lateral lobes.

In 1 of the cases a roentgenogram taken farther laterally showed the lateral lobes with the middle lobe located between them.

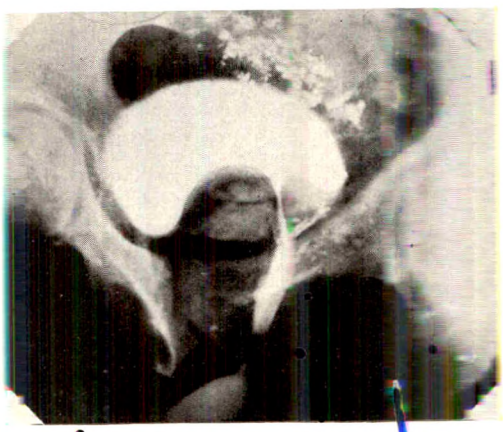


Fig. 16. Ventral or distal lobe (profile roentgenogram). Quite large intravesical growth of a tumor nodule. Extreme enlargement of the prostatic urethra.

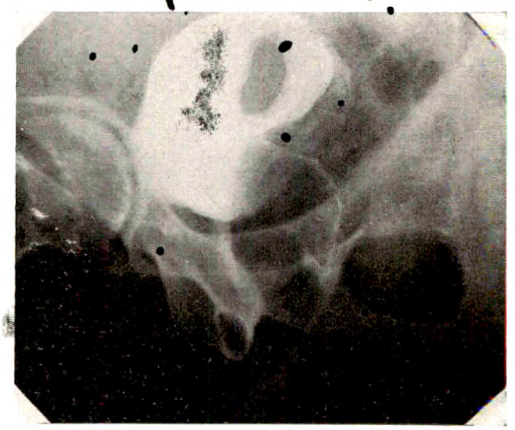


FIG. 17. Ventral or distal lobe (profile roentgenogram). Tumor growth partly intravesical and partly subvesical. Image of the prostatic urethra with the concavity forward. The subvesical part of the tumor growth reaches the membranous urethra, creating angular interstices, dangerous on instrumental examination. The roentgenogram shows the lacunar image of the accompanying bladder tumor (papilliferous cancer).

In the ventral or distal lobe, rarely seen, the "sphincter" is turned upward, the tumor nodule protruding into the base in front of the "sphincter" and with a volume that varies with the stage of growth (Fig. 16).

The image of the prostatic urethra, widened, elongated and deformed, sometimes shows a protrusion of the tumor into the lumen of the urethra (Fig. 17).

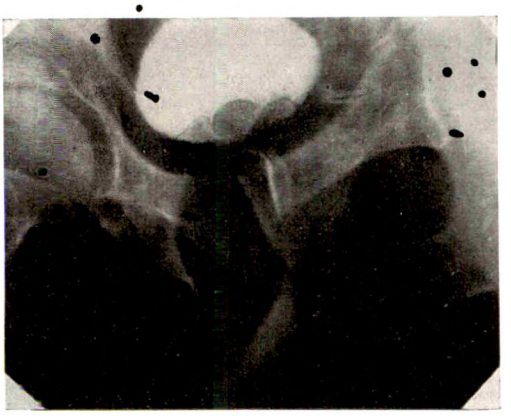


FIG. 18. Lateral lobes and middle lobe (profile roentgenogram). Intravesical protrusion of tumor nodules growing into the bladder. Angulation and enlargement of the prostatic urethra.

The protrusion corresponding to the edge of the posterior commissure of the neck projects more or less into the base, depending on the volume and growth of the ventral lobe. In total adenoma the lateral lobes in the profile roentgenogram project more or less into the base with equal or unequal growth of the two sides, the "sphincter" is turned upward, the image of the urethra is not elongated in the beginning but becomes increasingly elongated, broadened and deformed as the adenoma nodules increase in size (Fig. 18–28).

Sometimes the membranous urethra it-

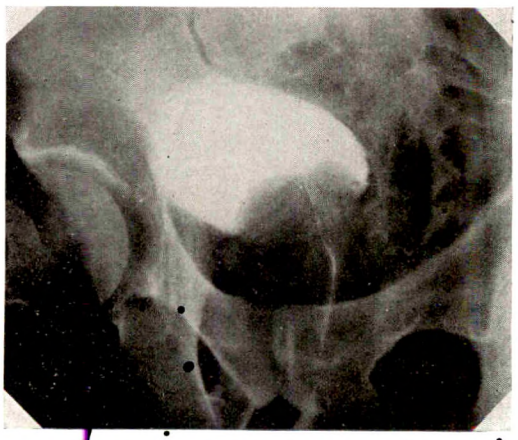


FIG. 19

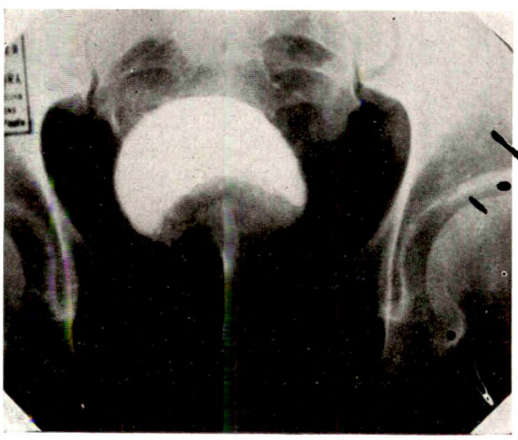


FIG. 20

FIG. 19. Total adenoma (profile roentgenogram). Intravesical growth of tumor. The prostatic urethra elongated and enlarged, the "sphincter" in the upper part of the tumor.

FIG. 20. The same case as Figure 19 (frontal roentgenogram).

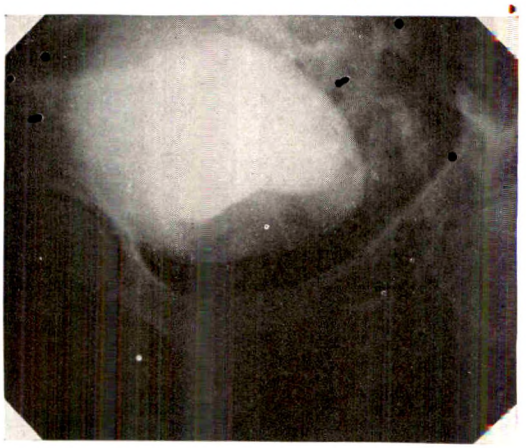


Fig. 21

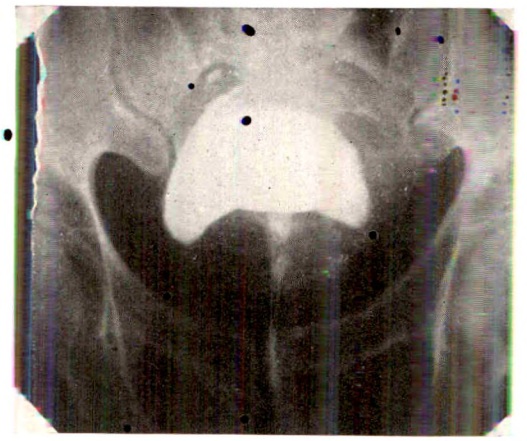


Fig. 22

Fig. 21. Total adenoma (profile roentgenogram). Intravesical protrusion of the tumor, but greater subvesical development.

Fig. 22. The same case as Figure 21 (frontal roentgenogram).

self becomes deformed with the subvesical growth of the adenoma and shows interstices which make instrumental examination dangerous; and in advanced stages the protrusion of the tumor reaches to the terminal part of the bulbar urethra.

In the frontal roentgenogram the image of the tumor protrudes into the base, the size varies depending on the stage of development and it may be subvesical or intravesical or partly subvesical and partly intravesical.

The image of the prostatic urethra is broadened, elongated and deformed, or is represented by a narrow thread of contrast medium due to the filling of the lumen by the tumor masses.* The roentgen appear-

* In the neighborhood of the "internal sphincter" it may bifurcate, showing that the contrast medium passes into the interstices of the tumor.

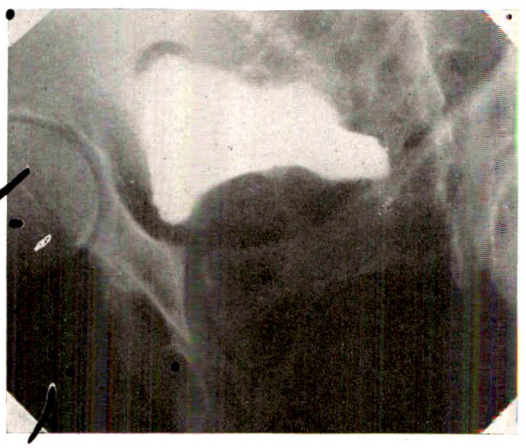


Fig. 23

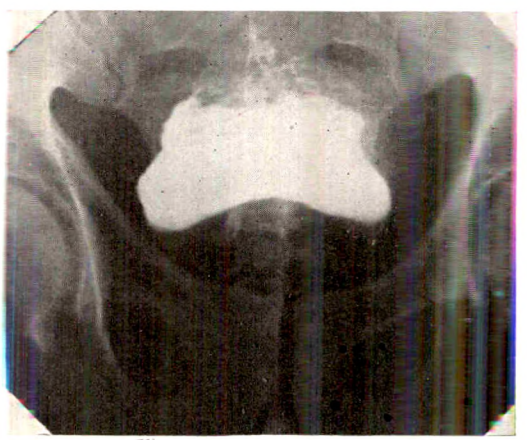


Fig. 24

Fig. 23. Total adenoma (profile roentgenogram). Though the tumor protrudes into the bladder cavity, the greater part of the growth is subvesical.

Fig. 24. The same case as in Figure 23 (profile roentgenogram). The cystogram shows signs of paresis of the bladder (attacks of retention) at the apex of the bladder.

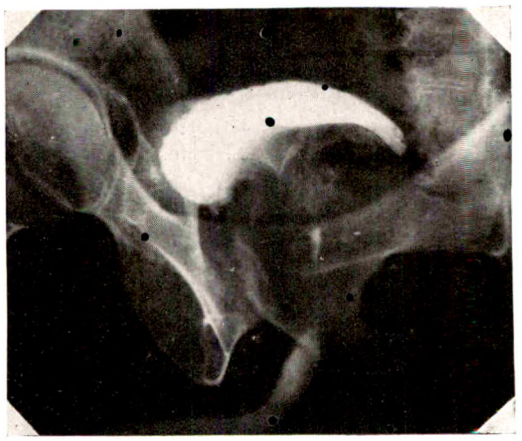


FIG. 25

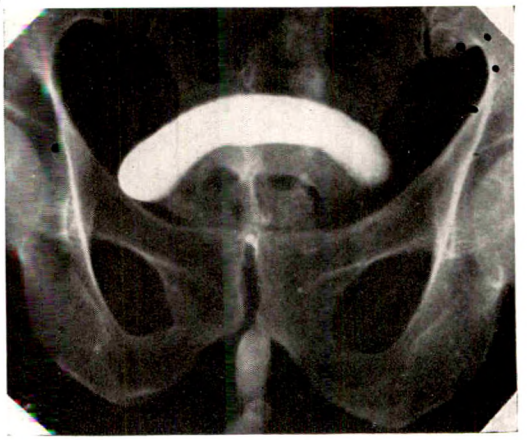


FIG. 26

FIG. 25. Total adenoma (profile roentgenogram). Large tumor with both intravesical and subvesical growth. The tumor in its growth has obstructed the prostatic urethra. The contrast medium has passed into the interstices in the tumor nodules.

FIG. 26. The same case as in Figure 25 (frontal roentgenogram).

ance depends, however, on the stage of development of the tumor.

In the 18 cases of this form of adenoma examined, we demonstrated the different stages of development from small adenomas of the lateral lobes to total adenomas and those of excessive development, some even with changes in the membranous and bulbar urethra. In many of these examinations the outline of the bladder showed changes caused by the adenoma, such as pulsion diverticula, true diverticula and a lacunar image corresponding to the bladder tumor (Fig. 17, 27, 29 and 30).

Findings in Urethrocystography. Though these unusual examinations were ordinarily carried out by skilled technicians there were a number of occurrences which deserve attention:

(*a*) Slight breaks in the mucosa of the

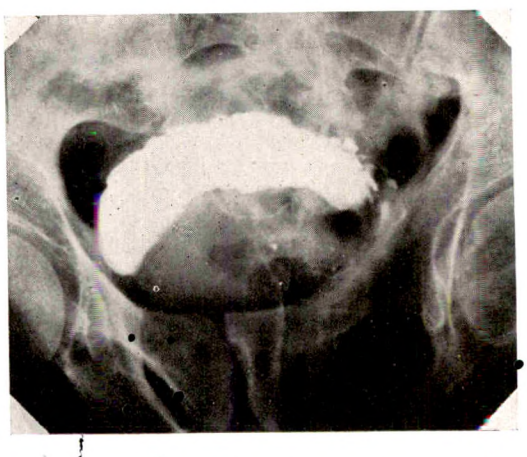


FIG. 27

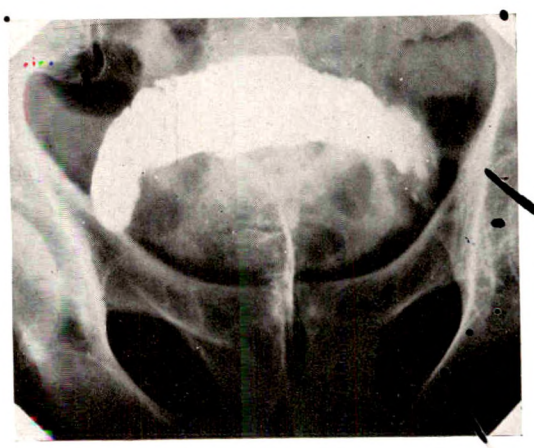


FIG. 28

FIG. 27. Total adenoma (profile roentgenogram). Tumor of very large size. Intravesical and subvesical growth. Great elongation of the prostatic urethra which is filled with tumor nodules.

FIG. 28. The same case as in Figure 27 (frontal roentgenogram). The cystogram shows numerous pulsion diverticula.

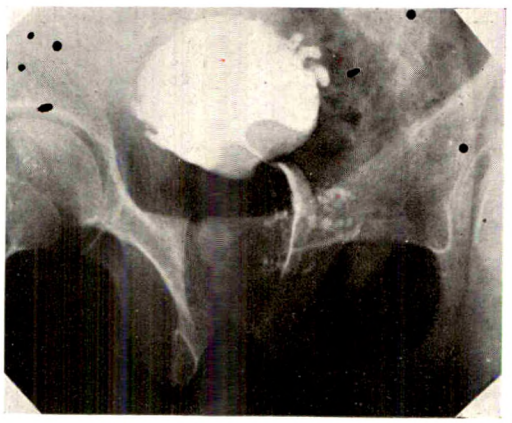


Fig. 29. Total adenoma (profile roentgenogram). Tumor with subvesical growth. Angulation, enlargement and elongation of the prostatic urethra. Prostatic glands dilated. Neck shows typical image of hypertonia of the sphincter. The cystogram shows pulsion diverticula.

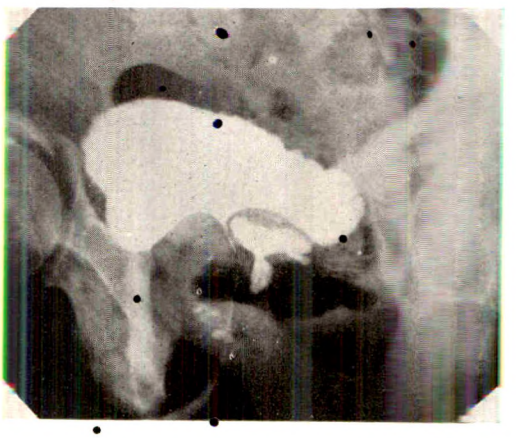


Fig. 30. Total adenoma (profile roentgenogram). Tumor of subvesical growth with abscess cavities. Part of the tumor projects into the bladder cavity.

prostatic urethra from distention during the filling. The patient passed contrast medium stained with blood. The mucosa, congested from stagnation or inflammation, suffered ruptures of the small vessels which made them bleed.

(*b*) Obstruction of the penetration of the contrast medium into the prostatic urethra when it was obstructed by growth of the tumor masses into it.

(*c*) Subadenomatous fissuring and loss of blood when a subvesical adenoma projected into the membranous urethra and enlarged it. The wave of liquid distended the interstices and fissured them.

(*d*) Fissuring of the bulbar cul-de-sac and loss of blood from its dissection in large subvesical adenomas.

(*e*) Even rupture of the bulbar cul-de-sac, involving the bulbar vein with penetration of contrast medium into the pudendal vein, reflux by the dorsal veins of the

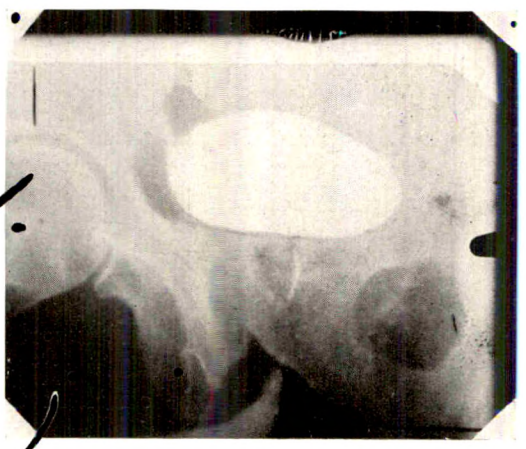


Fig. 31

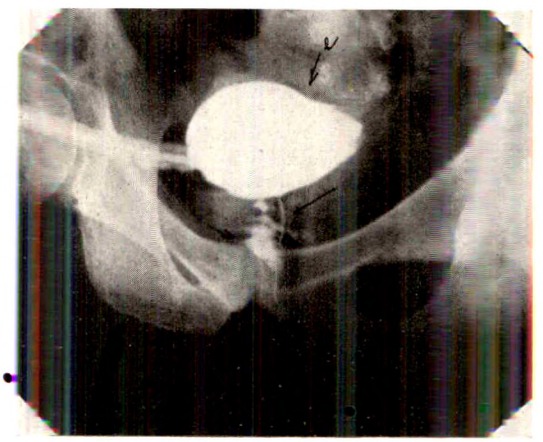


Fig. 32

Fig. 31. Congenital atrophy of the neck (profile roentgenogram). Atrophic prostate. (Plane image of the neck.)

Fig. 32. Sclerosis of the neck (inflammatory). (Profile roentgenogram.) Chronic prostatitis, stricture of the urethra, perineal fistulas. Suprapubic drainage (plane image of the neck).

penis and the dorsal emissary veins, penetration into the internal pudendal vein and impregnation of Santorini's plexus (urethrovenous reflux). Danger of hemorrhage, embolism, infection and generalized sepsis.

(f) Reactivation of latent inflammatory processes in the prostate and vesicles as well as reactivation of attacks of pyelonephritis in prostatics with urinary infection.

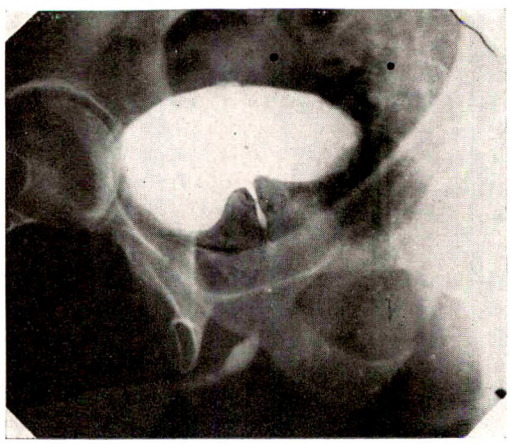


FIG. 33

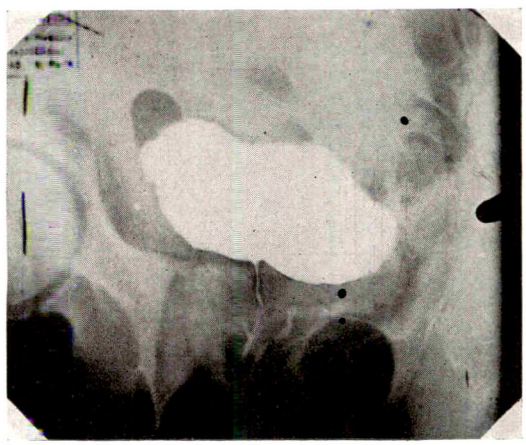


FIG. 34

FIG. 33. Hypertonia of the sphincter (profile roentgenogram). Chronic adnexitis with subacute exacerbations (typical fish-mouth image).

FIG. 34. Fibrosis of the neck (profile roentgenogram). Hypertrophy of the trigonal muscle. (Typical image.)

Among 31 patients with adenoma of the prostate on whom urethrocystography was done we observed the following:

Bleeding of the urethra from rupture of the mucous membrane	6
Reactivation of latent inflammatory processes of the prostate and vesicles	1
General depression after the examination	2
Reactivation of attacks of pyelonephritis in prostatics with urinary infection	2
	13

Among the remaining 18 patients examined there were no special findings. And particularly in nearly 100 cases of urethrocystography (stricture, ectasias of the neck, adenoma, etc.), there was never any urethrovenous reflux.[7,8]

Contraindications. There are, however, contraindications to urethrocystographic examination in the diagnosis of adenoma of the prostate which may be summarized as follows:

(*a*) When there is recent trauma resulting from instrumental examination, with or without loss of blood (danger of reflux and its sequels).

(*b*) In adenoma patients with acute or recent gonorrhea.

(*c*) In the course of subacute adnexitis (prostatovesiculitis) in patients with adenoma of the prostate.

(*d*) In the course of treatment of abscess of the prostate, particularly if it opens into the urethra, in patients with adenoma.

CONCLUSIONS

From the number of cases (31) examined of patients with adenoma of the prostate in all its clinical forms we conclude that urethrocystography as carried out by us is necessary and even indispensable in the examination of these patients for the following reasons:

(*a*) It gives the global or topographic appearance of adenoma of the pelvis.

(*b*) It shows accurately the forms of clinical manifestation (miniature form, median lobe, ventral lobe, lateral lobes, total adenoma) as well as the forms of its further development (vesical, subvesical).

(*c*) It shows the relation between an adenoma with subvesical growth and the parts of the membranous and bulbar urethra, giving a warning of the dangers of instrumental examination in these cases.

(*d*) It permits of differential diagnosis between adenoma and dysectasias of the neck (congenital atrophy, sclerosis, fibrosis of the neck and hypertonia of the sphincter (Fig. 31, 32, 33, 34).)

(*e*) It makes possible a better choice of the method of treatment for each of the clinical forms (suprapubic and perineal operations, endoscopic resection).

(*f*) Finally, it permits of verification of postoperative incidents and complications (structural deformities) which would retard clinical cure of the patients operated on, and shows recurrences of the tumor.

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METASTASIS TO BONE AS THE FIRST SYMPTOM OF CANCER OF THE GASTROINTESTINAL TRACT*

REPORT OF THREE CASES

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A REVIEW of the literature reveals that although metastasis to bone from cancer of the gastrointestinal tract occurs more frequently than is generally realized, cases in which the bone lesion produces the first symptom of these cancers are quite rare.

The 3 cases here presented sought relief from the symptoms caused by the bone involvement, none of whom had any important symptoms referable to the gastrointestinal tract. A biopsy revealed the true nature of the tumors, and in each instance the pathologist, Dr. A. R. Camero, suggested the probable source of the bone lesions. Gastrointestinal roentgen studies were made only after the pathological diagnosis of adenocarcinoma had been made and, in each case, the primary growth was found in the gastrointestinal tract: 1 in the stomach and 2 in the sigmoid colon. Metastasis to the regional lymph nodes and to the surrounding and neighboring organs is frequent, but judged by our own experience and by the records in medical literature, distant bone metastasis from cancer of the digestive tract is relatively uncommon so far as it is found clinically even after the primary lesion has been discovered. Many of these cases are undoubtedly missed because it is usually not practical to make a complete roentgen examination of the entire osseous system unless there is a suspicion or sign or symptom suggesting bone involvement. The pathologist, no doubt, also fails to recognize a number of these cases because the usual autopsy on patients dying from gastrointestinal cancer does not include a detailed study of the skeletal system.

Our 3 cases were discovered in a series of 186 patients with carcinoma of the gastrointestinal tract over a period of ten years (1932 to 1941): 13 in the esophagus, 75 in the stomach, and 98 in the colon and rectum.

Recently Buirge,[4] in a report on carcinoma of the large intestine, reviewed 416 autopsy records, finding 31 per cent of this number incorrectly diagnosed. Sixteen per cent of the cecal lesions were diagnosed appendicitis and 8.3 per cent as cholecystitis. Recent hemorrhoidectomies had been performed on 5.2 per cent of the cases, without discovering the primary lesion in the sigmoid or rectum. It is a well-known fact that the early symptoms of cancer of the colon may be of such a vague and insignificant nature, or even absent, that both the patient and the physician may be misled and the diagnosis made only after the occurrence of distant metastases. In a series of 391 autopsies on patients with primary neoplasms of the digestive tract, Turner and Jaffe[22] found 7.9 per cent with bone involvement. Rendich and Levy[18] report 6 unusual cases of bone metastasis, 2 of which were secondary to gastrointestinal carcinoma. These 2 cases are similar to my own in that the chief complaint was caused by the distant bone metastasis rather than the primary growth, and in 2 instances the secondary growths were found below the knee. Nisnjewitsch[17] reported 6 instances of bone metastasis, found at postmortem, in 57 cases (10.5 per cent) of primary carcinoma of the rectum. Mathews[14] found an incidence of 2.5 per cent of bone involvement from gastric cancer. Moore[15] found, in his study of 1,600 cases of carcinoma of the stomach, no evidence of bone involvement. Jenkinson,[10] in his series of gastrointestinal cancer giving rise to bone metastasis, found no tumors secondary to carcinoma of the stomach, but found a record of 21

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cases in the literature in which the metastasis occurred from different parts of the gastrointestinal tract. Warwick[24] reports metastasis to bone in 4 out of 176 autopsy cases of gastric carcinoma, or 2.2 per cent. Aufses[1] reviewed a series of 117 cases of rectal carcinoma and found 8 instances of bone involvement, or 6.8 per cent. In his series of 537 cases of carcinoma of the stomach, from Johns Hopkins Hospital, Copeland[6] reports 7 cases with bone lesions, or 1.3 per cent. Sutherland[20] states that 1.9 per cent of his 1,032 cases of malignant bone metastasis arose from primary growths in the stomach. Warren[23] found 5.7 per cent of bone involvement in his gastric cases, and also reported 2 cases with bone involvement in 156 cases of carcinoma of the colon. Willis[25] reports the incidence of skeletal metastasis from carcinoma of the stomach as approximately 5 per cent. In 1935, Kerr and Berger[11] reviewed the literature, finding 143 cases and added 5 of their own. In his series of 606 cases of gastric carcinoma, Lawton[12] reports only 3 with bone involvement. Clayton[5] in 41 cases of carcinoma of the esophagus, found 2 cases with bone metastasis. Ghormley and Valls[8] surveyed the records of cases in which a diagnosis of carcinoma of the gastrointestinal tract was made at the Mayo Clinic in the fifteen year period from 1922 to 1936. The growths occurred in the esophagus, stomach, duodenum, jejunum, colon, sigmoid, rectosigmoid, and rectum. The greater number of cases of metastasis to bone from carcinoma of the rectum and stomach, they thought, was due to the greater incidence of carcinoma in these organs. Only 1 case of bone metastasis from small bowel carcinoma was found. The accompanying table shows the distribution and location of bone metastases in their 43 reported cases (Table 1). The table indicates that most skeletal metastatic growths are in the bones of the trunk; that is, in the vertebrae, the pelvis and the ribs. In only 6 cases were long bones involved. This distribution would be expected because bone metastases from most types of carcinoma have a similar distribution. Most of the reported cases show the majority to be osteolytic although osteoblastic lesions also occur. When the metastases are osteoblastic and associated with carcinoma of the rectum, primary prostatic involvement must be suspected. The destruction of bone and bone marrow may be very extensive, producing a blood picture strongly suggesting pernicious anemia. Markowitz[13] reports such a case in which the erythrocyte count was 1,010,000; hemoglobin, 15 per cent; color index, 1.1. The postmortem examination showed adenocarcinoma of the stomach

TABLE 1

DISTRIBUTION AND LOCATION OF BONE METASTASES IN 43 CASES (GHORMLEY AND VALLS[8])

Location	No.	Secondary Growths							Incidence of metastasis to bone for all carcinomata of the indicated organs (per cent)
		Location							
		Spine	Pelvis	Ribs	Long bones	Sternum	Scapula		
Esophagus	1			1				1	0.2
Stomach	12	6	3	5	2	1		17	0.2
Small bowel	1	1						1	
Colon	3	1	1	1				3	
Sigmoid	3	2		2	1			5	0.3
Rectosigmoid	4	3	2					5	
Rectum	19	6	8	1	3		1	19	0.5
Total	43	19	14	10	6	1	1	51	

with extensive bone metastasis. The first symptom in his case was pain in the legs and rapidly progressing anemia with no special gastrointestinal symptoms. Barclay[2] reports a case of colloid carcinoma of the stomach, the first symptom being a pathological fracture of the clavicle while turning in bed. No gastrointestinal study was done, there being no symptoms referable to this tract. Roentgen examination revealed a generalized carcinomatosis involving practically all of the bones of the body including the extremities. The site of the primary lesion was not determined. The erythrocyte count was 1,500,000; hemoglobin, 33 per cent; color index, 1. No gastrointestinal symptoms developed until shortly before death. Stein,[19] in his series of 1,005 cases of carcinoma of the digestive tract, reports 34 with bone metastasis. In this series he includes carcinoma of the pancreas, gallbladder and biliary ducts, one of which was the result of primary carcinoma of the small bowel. Fort[7] also reports a single case of bone metastasis from carcinoma of the small bowel. Tilling[21] reported a case in which a metastatic growth in the humerus was the first sign of gastric cancer. Müller[16] studied the records of 12,730 postmortem examinations performed at Basle Pathological Institute from 1817 to 1905. His findings are shown in Table II. The high incidence (12.09 per cent) is in part explained by the fact that his studies were limited to postmortem material.

TABLE II
POSTMORTEM FINDINGS

Location of Primary Carcinoma	No. of Cases	Cases with Metastasis to Bone	
		No.	Per cent
Stomach	309	8	2.58
Esophagus	101	7	6.93
Rectum	57	6	10.52
Gallbladder	56	2	3.57
Pancreas	19	4	21.05
Liver	15	3	20.00
Pharynx	10	2	20.00
Total	567	32	Av. 12 09

Müller studied the records of 12,730 postmortem examinations performed at Basle Pathological Institute from 1817 to 1905 and found 567 cases of carcinoma of the gastrointestinal tract.

The principal recognized methods of spread of malignant metastasis are: lymph and blood borne emboli, lymph and blood vessel permeation and various combinations of these routes. Gray[9] has recently shown that the spread of cancer by lymph vessel permeation is less common than is generally supposed. Batson[3] has demonstrated by experimental work on the cadaver and monkeys the probable route by which many distant metastases occur. We have all seen metastatic tumors appear in locations that do not seem to be in line of direct spread from the primary focus. Batson injected radiopaque material into the dorsal vein of the penis. The material did not enter the inferior vena cava but progressed up the spine. Many intercostal veins were filled as well as the veins of the pelvis. When the amount of the injection was 200 cc., the material reached the base of the skull and entered the cranial cavity. It also extended down into the thigh, through fine straight vessels. Valves prevented the filling of the larger veins of the thigh. These experiments were repeated on living monkeys with the same distribution of the injected material. Batson has demonstrated that the entire system of epidural and vertebral veins has a free anastomosis at each spinal segment with the veins of the thoracic and abdominal cavities. It is a system of veins without valves, except in minor connecting channels, and with very low pressure. With every compression of the trunk such as occurs by coughing, lifting and straining, the intratruncal pressure is raised to a sufficient height so that blood flows, not into the inferior vena cava, but into this vertebral system of veins. He believes that many distant metastatic lesions may occur through these veins without involving the portal, pulmonary or caval system. This method of spread could very well account for the distant isolated bone lesions produced in our cases: one from the sigmoid colon to

the skull; one from the sigmoid to a rib, and one from the stomach to the left fibula. The routes and methods of dissemination of malignant emboli are so varied and extensive that it is not possible to anticipate the probable location of their development. Very distant lesions may be produced without any demonstrable intervening metastatic involvement. For these reasons it is practically never possible for the surgeon or anyone else to determine with any degree of certainty the curability of cancer of the gastrointestinal tract regardless of its duration, size, location, mobility or any other of its many varied qualities or characteristics.

CASE REPORTS

CASE 1. A female, aged fifty-seven, white, well nourished, was referred to the Misericordia Hospital on October 11, 1939, as an ambulatory patient for the removal of a "sebaceous cyst" from the scalp. About four weeks ago she noticed a sore spot in the left temporal region. A lump developed in this area which gradually increased in size, until now there is a rather hard, fixed, reddish tumor about 2 cm. in diameter, located at the hair line in the left frontal region. Under local anesthesia an incision was made and immediately it was recognized by the surgeon that this was not a "cyst," but a tumor which had already involved and destroyed a considerable area of the frontal bone. Most of the soft tissue mass was removed and sent to the laboratory.

Pathological Report. "The histopathologic picture here seen is typically that of adenocarcinoma and from its appearance it is apparently a metastatic lesion. In the better differentiated portions the appearance of the epithelial tumor cells suggests the large bowel as the primary site but we cannot be sure of this."

The patient was then admitted to the hospital for extensive studies. She has been feeling entirely well and had no complaint except for an occasional pain in the region of the recently excised tumor. Her appetite has been good and she has had no gastrointestinal complaints of any kind; no abdominal pain, nausea or vomiting. Bowel function has been entirely normal.

Physical examination revealed nothing of importance except the lesion in the left frontal region.

Laboratory Findings. Erythrocytes, 4,250,000; hemoglobin 84 per cent; leukocytes, 7,500. Urine negative for casts, sugar and albumin.

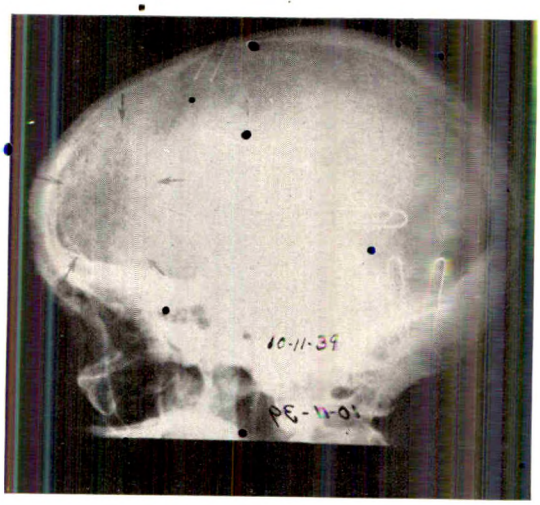


FIG. 1. Case 1. Roentgenogram made through bandages immediately after the attempt to remove the supposed 'sebaceous cyst," showing the destructive metastatic lesion in the left frontal bone. October 11, 1939.

Roentgen Findings. In a roentgen examination made on October 11, 1939, the skull shows an irregular area of bone destruction, approximately 5 cm. in diameter, involving the left frontal bone. The nature of this lesion cannot be determined from this study, but the irregular margin associated with the soft tissue tumor suggests possible metastatic malignant disease (Fig. 1). The neck, chest, spine and pelvis are normal in appearance. Gastrointestinal examination shows the stomach normal. In the colon there are many large, saccular diverticula and a constant constriction in the sigmoid, shown on several roentgenograms. This slight deformity could readily be produced by the diverticula and associated colitis, but if the abdomen is opened this area should be carefully examined for a possible small annular carcinoma. From the appearance alone an inflammatory lesion is favored, but in the light of the biopsy, carcinoma must be considered. Several roentgenograms were made of the filled colon on two different occasions and several after the double contrast enema, all of which show the same constriction (Fig. 2).

Operation (November 8, 1939). Under spinal anesthesia the sigmoid was exposed and freed. A small hard mass was identified and about 12 cm. of the sigmoid was resected. An end-to-end anastomosis was performed. The liver was then palpated and found to be studded with many



FIG. 2. Case 1. Roentgenogram of colon shows persistent constriction near the middle of the sigmoid, proved by resection to be a small adenocarcinoma. Multiple large diverticula are also present.

nodules about the size of an olive. A small incision was made in McBurney's point, and a cecostomy performed.

Gross Pathological Description. Specimen consists of 11 cm. of resected sigmoid. At about the middle of this specimen there is partial constriction of the lumen. On the mucosal aspect a small circular ulcerative tumor is noted which measures 1.5 cm. in diameter. This tumor has firm, everted margins and its base is quite firm. Several saccular diverticula are also noted, the orifices of which are quite narrow.

Microscopic Examination. The tumor is made up of large irregular masses of anaplastic epithelial tumor cells, arranged in very haphazard glandular formations, and separated by abundant dense fibrous connective tissue stroma. The cells show the various cytologic features of rapid growth, including some mitoses. Tumor tissue is seen to extend deeply into all layers of the bowel wall. Diagnosis: primary adenocarcinoma of the sigmoid.

The patient made a good recovery from the operation and remained fairly well until January, 1940, when vomiting, loss of appetite, loss of weight, jaundice and abdominal distention developed. From November 6, 1939, to January 25, 1940, the patient received 4,165 roentgens (125 kv., 6 mm. Al filter) over the skull lesion, but the tumor continued to increase in size until it involved an area 10 cm. in diameter, with almost complete destruction of the left half of the frontal bone. Other small scattered destructive lesions were found in the skull at this time (Fig. 3 and 4). The patient died on April 15, 1940, a little more than five months after the operation.

CASE II. This patient, white, female, aged seventy, was admitted to Misericordia Hospital November 22, 1940, because of pain and swelling in the left leg which began about three months ago. She consulted a physician soon after the onset of pain and was advised to stay

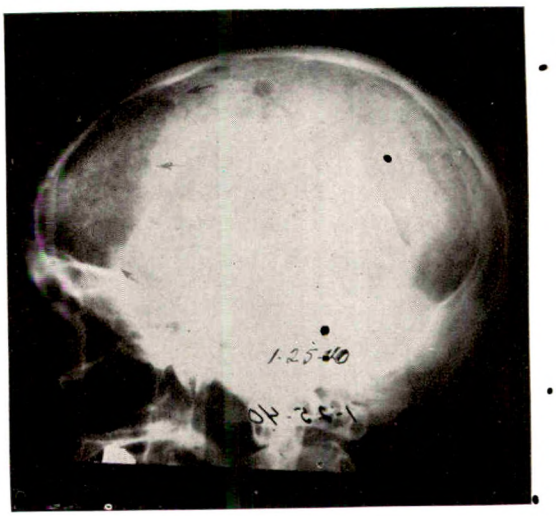


FIG. 3. Case 1. Left lateral view of the skull showing increase of destruction in the left frontal bone. January 25, 1940.

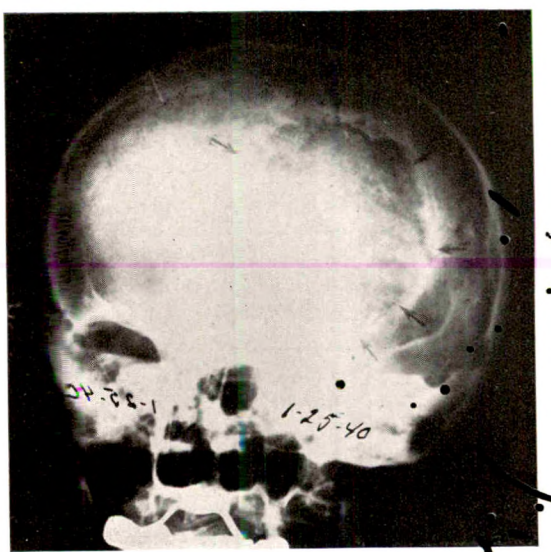


FIG. 4. Case 1. Oblique frontal view also made on January 25, 1940, showing large osteolytic lesion in left frontal bone.

off her feet as much as possible. She obtained no relief and was then admitted to the hospital. General health was good. She had an appendectomy performed at another hospital three months ago.

Physical Examination. Head, neck and chest grossly negative. Abdominal scar of recent appendectomy; no areas of tenderness or rigidity; no organs or masses palpable; abdominal skin lax and atrophic. *Extremities:* Patient complains of pain in left leg and over the tarsal bones of the left foot. There is present a palpable tumor in the outer portion of the left leg, just below the knee, and popliteal pulsation. No loss of temperature and varicose veins are present from the knee to foot.

Roentgen Findings. The left leg and ankle show a destructive lesion involving the upper 8 cm. of the fibula, except for a small portion of its upper extremity. There is complete destruction of the bone in this area. The bone immediately adjacent to the lesion is normal in structure. We have, therefore, a purely osteolytic lesion with no tendency to bone production or periosteal reaction. The appearance is strongly suggestive of solitary myeloma (Fig. 5). Roentgenograms of the skull, chest, spine, pelvis and right knee show no similar lesions. On the basis of these findings the upper third of the fibula was resected.

Laboratory Findings. Urinalysis negative for sugar, casts, albumin and Bence-Jones protein. Blood Wassermann and Kahn reactions negative. Erythrocyte count, 3,720,000; hemoglobin, 67 per cent; leukocyte count, 6,000; serum calcium, 11.

Microscopic Examination. Tissue submitted shows the characteristic picture of adenocarcinoma. After the pathological report a gastrointestinal roentgen examination was made and this shows a large, irregular filling defect, about 2½ inches in extent, involving the cardiac end of the stomach. There are also present two sharply defined transparencies along the greater curvature, suggesting polyps (Fig. 6). There can be little doubt that the metastatic lesion in the left fibula developed from the primary carcinoma of the stomach. Metastasis occurring below the elbow and knee, from primary carcinoma in any organ of the body, is uncommon but from carcinoma of the stomach such lesions must be very rare.

CASE III. This patient, a white, fairly well nourished female, aged fifty-one, was admitted to the surgical ward of Misericordia Hospital, November 25, 1940. About two months ago the patient first noticed a lump in the left side of her neck. She has lost about 12 pounds in weight in the past four years. During the past five years

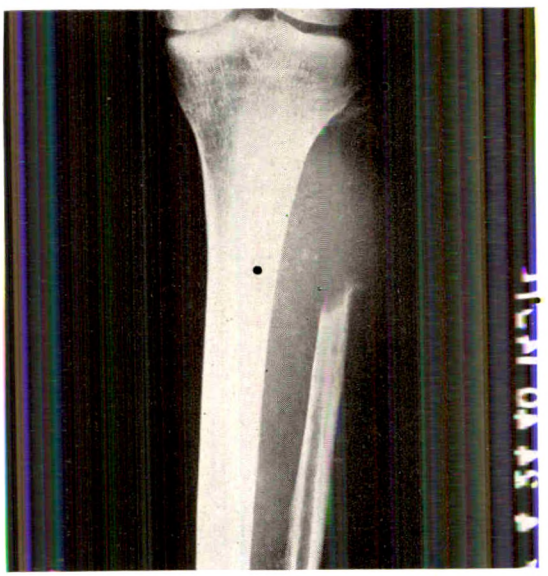


FIG. 5. Case II. Upper two-thirds of left leg showing metastatic osteolytic lesion in upper fibula.

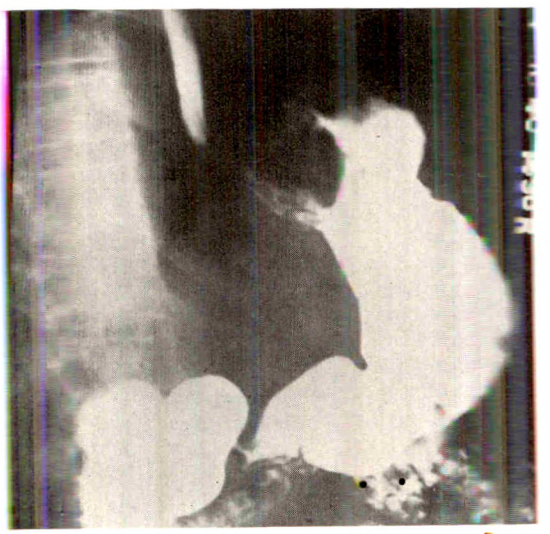


FIG. 6. Case II. Large primary adenocarcinoma in cardiac end of stomach and two polyps along greater curvature.

she has passed blood in her stools several times. No urinary symptoms or gastric distress. Erythrocyte count, 4,750,000; hemoglobin, 74 per cent; leukocyte count, 8,600.

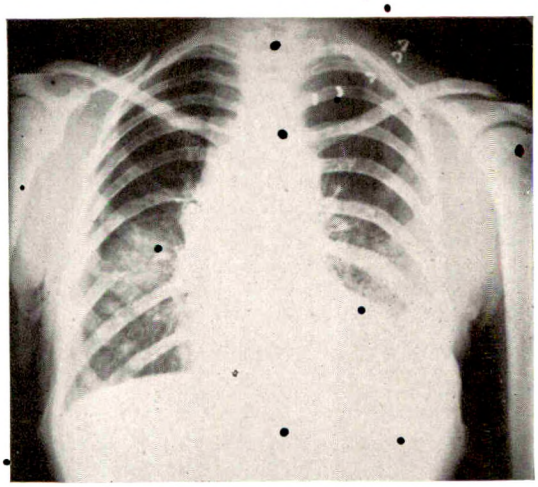


FIG. 7. Case III. Metastatic lesion in the eighth right rib, posteriorly, and soft tissue tumor which replaced the destroyed bone. Disease also in left base.

The patient presents a nodular mass in the left supraclavicular region about the size of a plum. The clinical impression was benign adenoma of the thyroid gland; lymphoma; lymphosarcoma.

On November 26, 1940, an incision was made over the mass and several large lymph nodes were identified. These were removed and sent to the laboratory. The same afternoon a roentgenogram of the chest was made which shows a destructive lesion involving about 3 inches of the eighth right rib posteriorly. The destroyed portion of the rib is replaced by a soft tissue mass. The nature of the osteolytic lesion cannot be determined from this examination (Fig. 7).

Microscopic Examination. Examination of the firm, matted lymph nodes shows the normal architecture almost completely destroyed and replaced by tumor elements consisting of large, irregular masses of anaplastic epithelial tumor cells arranged in haphazard glandular formations, and separated by abundant dense fibrous tissue stroma. The histopathologic picture seen here is typically that of adenocarcinoma, apparently metastatic. The appearance of some of the tumor cells suggests origin from the stomach or other part of the gastrointestinal tract. Barium studies were then made. The stomach and duodenum appear normal. The colon shows a persistent irregular filling defect at the junction of the descending and sigmoid portions, involving an area about $2\frac{1}{2}$ inches in extent, reducing the lumen to about 0.75 cm. in diameter. The appearance is strongly suggestive of carcinoma of the sigmoid (Fig. 8). Although in this case the patient sought relief from the enlargement in the left side of the neck, the metastatic lesion in the eighth right rib was found before the primary tumor in the sigmoid was suspected or discovered.

DISCUSSION

The 3 cases here presented emphasize the difficulties in making a diagnosis of cancer of the gastrointestinal tract while it is still operable. In Case III an earlier diagnosis might have been made if the bleeding from the rectum, which occurred at intervals over a period of five years, had been carefully investigated. In Cases I and II, however, there was nothing in the histories or examinations to even suggest the presence of deep-seated malignant disease. In Case III the metastatic lesion of the rib was absolutely painless at the time the chest roentgenogram was made. Pain and tenderness, however, did develop in the back and lower right chest eleven days after dissection of the enlarged glands in the neck. The bone lesions in the other 2 cases were moderately painful and showed definite tumor formation. Cancer of the digestive tract was not suspected in any of the 3 cases, and the probable location of the primary growth was indicated by the microscopic examinations.

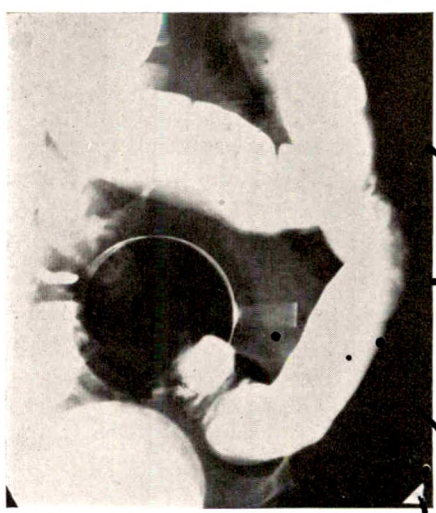


FIG. 8. Case III. Primary adenocarcinoma of the sigmoid. (Circle over lesion is due to aluminum compression cup.)

The literature reveals a considerable number of cases of bone metastases from gastrointestinal carcinoma, but only a few cases in which the primary tumor was located from the study of the metastatic lesion in the bone. There can be no doubt that a considerable number of these cases go unrecognized because few, if any, roentgenologists routinely roentgenograph all the bones of the body in conjunction with barium studies. At postmortem, too, it is not practical to study the entire skeleton in great detail in the absence of a definite bone lesion. The size of the lesion has little or nothing to do with determining the presence or absence of metastasis. In Case 1, the growth in the sigmoid was only 1.5 cm., and the metastatic deposit in the skull 5 cm. in diameter.

CONCLUSIONS

(1) Cancer may be present in the gastrointestinal tract for a considerable time without producing significant digestive disturbances.

(2) Bone metastasis from cancer of the digestive tract is more common than is generally realized.

(3) Bone metastasis as the *first sign* of gastrointestinal cancer is quite rare.

(4) More thorough skeletal roentgen and postmortem examinations would undoubtedly reveal a greater number of cases.

(5) Metastatic lesions in bone may attain considerable size before producing pain or any other symptom.

(6) The three cases here presented show purely osteolytic lesions.

(7) The route by which distant metastasis frequently occurs is probably through the "vertebral veins" (Batson).

(8) In our single treated case, roentgen therapy had no effect on the superficial soft tissue tumor or the bone lesion. The destruction of the skull and tumor of the scalp continued to progress until death.

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DISCUSSION

DR. GEORGE E. PFAHLER, Philadelphia. Many years ago the Emperer of China called together his greatest philosophers and asked them to formulate in the fewest possible words a philosophy that would fit all occasions, and finally after much deliberation they came back with three words or a sentence of three words—"It will pass." You will see that those three words do cover everything that we can think of.

Ross Golden has given us a gem in the introduction to his fine piece of work, the Nelson loose-leaf diagnostic book, when he said that the first essential in making a diagnosis is to think of it. In other words, you must think of the diagnosis before you can possibly make it. We can therefore summarize, as a philosphical point, our troubles in diagnosis by again boiling down the advice into three words, "Think of it." That is what Dr. Bertin has emphasized this morning in presenting these three cases. He has made us think of the possibility of a lesion that is found in an unusual place as being due to a metastatic cancer secondary to the gastrointestinal tract.

It is true that not many cases are recorded. I think Dr. Bertin has found only two other cases of this type in the literature. I have been doing gastroenterology for thirty-seven years. I have never come across any such case preceding these three which Dr. Bertin and I observed together at the Misericordia Hospital, but there probably are other cases and if we get into the habit of thinking of this, we will probably find others.

Dr. Bertin has referred to a case in which the first symptoms were due to metastatic nodules in the neck. That reminds me of a case that was brought to the Graduate Hospital with a recurrence after two operations for adenocarcinoma on the left side of the neck. The primary lesion was not found. I did not think of a gastrointestinal origin, and by very careful examination—and this patient had been examined repeatedly by the laryngologist—no lesion was found, but by palpation I found a small lesion, less than 0.5 cm. in diameter in the tonsil, which was found to be the primary lesion in that case. So we must think of these various possibilities, and it isn't only the gastrointestinal tract that we must think of, but Dr. Bertin has added this as another real contribution to make us think of hunting more thoroughly for the primary lesion.

The unusual distribution of the metastatic carcinoma as described in the paper by Dr. Bertin can probably be explained on the basis of distribution through the valveless venous channels as described by Batson, and as stated in his remarkable demonstration in our scientific exhibit made for this Society a year ago. He has shown that metastatic cancer cells can enter the venous channels and be distributed along the spinal veins as far as the head, and also into the venous channels of the lower extremities. Batson has illustrated the mode of dissemination of disease by calling our attention to the metastasis as found in biliary tuberculosis in which the disease is scattered through the arterial blood streams and distributes the multiple foci at random. In erysipelas, organisms invade a plexus of lymph vessels and multiply. As a result, the primary lesion enlarges, colony-like, from its margins. In tuberculous adenitis, the organisms spread in lymph channels to multiply in the regional lymph nodes and lymph node chains. These methods of distribution of metastatic cancer are also found and have been well recognized in the past, but Batson has described the fourth method by which the cancer cells are distributed and disseminated through the valveless venous system. It is entirely likely, therefore, that this is the explanation of the distribution of the lesion that occurred in the frontal bone, the one in the leg, while in the remaining or third case the deposit was probably distributed directly through the lymph channels, and implanted in the supraclavicular region.

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NEUROFIBROMATOSIS OF BONE*

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NEUROFIBROMATOSIS (Recklinghausen's disease[22]) was originally thought to be a disease of the peripheral nervous system characterized by the development of cutaneous nodules and subcutaneous tumors arising from the peripheral nerves. Since von Recklinghausen's publication in 1882 numerous additional cases have been reported in the literature which have altered the conception of this disease. It is now generally considered to be hereditary in character (Preiser and Davenport;[21] Gardner and Frazier;[8] Sharpe and Young[23]). Among the case reports which have brought to light new aspects of this disease are included: pigmentation of the skin (Thomson;[27] Leader and Grand[13]), mental deficiency (Gordon;[9] Yakovlev and Guthrie[30]), involvement of the central nervous system (Cushing;[4] Aegerter and Smith[2]), congenital developmental defects (Thomson;[27] Ducroquet;[6] Ducroquet and Cottard[7]), and alterations in the skeleton (Adrian[1]; Gould[10]; Lehman and Brooks;[15] Barber[3]).

It is the purpose of this paper to present several additional cases of neurofibromatosis, one of which presents an unusual manifestation of this disease in the bones.

The diagnosis of neurofibromatosis is usually not difficult. Stout, Laidlaw and Haagensen[26] described the characteristic feature of Recklinghausen's disease as a proliferation of the tissues composing the peripheral nerves, including both the axis cylinders and their sheaths. The overgrowth may take place along the course of the nerve, produce a general thickening of the nerves or may involve their peripheral terminations. Bone changes, which are found in 7 per cent of cases (Hodges, Pfhemister and Brunschwig[11]), combined with the characteristic pedunculated soft tissue tumors and coffee colored splotches on the skin make the condition unmistakable.

SKELETAL CHANGES

Early reports of bone changes in neurofibromatosis are found in the extensive literature of this condition (Adrian[1]). Basing his opinion primarily on the alterations in the bones, Stahnke[24] was the first to suggest that the affection is a deep seated systemic disease. Weiss[29] had previously called attention to the common occurrence of scoliosis with neurofibromatosis.

In 1924 Lehman and Brooks[15] classified the bone changes in Recklinghausen's disease as (1) scoliosis, (2) abnormalities of growth of the individual bones associated with elephantiasis, and (3) irregularity in the outline of the bones ranging from central and subperiosteal cysts to pedunculated subperiosteal tumors. The authors describe as the fundamental cause of the bone changes the development of a neurofibroma in one of the periosteal nerves. As the tumor enlarges, pressure is exerted on the bone cortex causing erosion and pit formation. These depressions are filled with neurofibromatous tissue. In the roentgenogram they sometimes take on the appearance of cysts and at times a thin shell of cortex covers the fibrous tissue. Five of the 7 cases reported showed roentgen evidence of bone involvement. Biopsies were obtained in 2 of the cases but in neither instance could any actual infiltration of the bone substance by neurofibromatous tissue be demonstrated.

Gould[10] examined microscopically portions of the skeletons of 5 cases of neurofibromatosis. One of these cases, which presented typical changes of osteomalacia, showed also characteristic microscopic findings. A second case showed similar changes although somewhat less marked. There was

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no evidence of invasion of the bone by neurofibromatous tissue. The remaining 3 cases showed no abnormalities in the bones. Increased porosity and softening of the bones were described by Lehman and Brooks[15] which they attributed to the hyperplastic changes in the lymphatics associated with the growth of the neurofibroma. Subsequently Lehman[14] reported a case in which a biopsy removed from an enlarged innominate bone showed a marked osteoporosis with large haversian spaces and a thin cortex. He was unable, however, to demonstrate neurofibromatous tissue within the bone. The bone cut with great ease at the operation. The hypothesis was advanced that with the increased porosity and softening of the bone there is a growth in the length of the bone which is distributed throughout its entire extent instead of being confined to the region of the epiphyseal cartilage. Conversely, the presence of neurofibromatous tissue growing in such a way as to destroy the epiphyseal cartilages results in an abnormally short bone. Nørgaard,[19] however, is of the opinion that differences in the length of bones can be accounted for on the basis of an embryonal defect. Moore[18] finds strong evidence that overgrowth is the result of a segmental relationship between the affected nerve and the bone. Spontaneous fractures of bones and the development of a pseudarthrosis have been described in bone which showed osteoporosis.

Scoliosis is found frequently in neurofibromatosis. Miller[17] reported it as a finding in 43 per cent of cases. He describes the deformity as fairly characteristic, consisting of a kyphoscoliosis which differs in several important details from the usual type of kyphosis or scoliosis. The usual site is the lower portion of the thoracic spine. The kyphotic element was found to be strongly predominant while the scoliosis is slight and presents a moderate degree of rotation. The cause of the scoliosis has been attributed to bone softening (Gould[16]), to neurofibromata of spinal nerves (Lehman and Brooks[15]), to asymmetry of the extremities (Nørgaard[19]), and to developmental defects in the vertebrae (Moore[18]).

The following case illustrates a striking deformity of the thoracic spine which apparently has its origin in a congenital defect.

CASE 1 (Unit No. 388828). E. K., a white baby, was first admitted to the hospital March 3, 1910, at the age of four and a half months when his mother noticed an angulation of the ribs on the right side near the attachment of the spine. She attributed this to an injury sustained when the child was dropped by his nurse. At the age of six and a half months cord-like masses were observed in the intercostal spaces between the seventh, eighth, ninth and tenth ribs on the right side. A diagnosis of neurofibromatosis was made at this time. At the age of four years he had a series of nose bleeds. By the time the patient reached his fifth birthday his spine began to show an angular deformity which increased gradually, reaching its maximum at about seventeen years. A pedunculated lump developed below the spinal deformity which increased in size until it reached the dimensions of a football. On several occasions this lump was injured accidentally, causing hemorrhage within it, which was complicated by a secondary infection. This required hospitalization for incision and drainage.

The patient's father had numerous pedunculated tumors of the skin of the arms, legs and trunk, which on examination were found to be neurofibromata. Roentgenograms of his bones showed nothing unusual. A brother had several irregular brownish areas of pigmentation on the skin on the back of his neck.

Physical examination showed a marked dorsal kyphosis of the spine which forms a pointed lump with the vertex at about the tenth thoracic vertebra. The vertebrae are rotated toward the right so that the spinous processes point to the left a little anterior to the lateral plane. From the spine the ribs extend in a distorted manner forward to form a thoracic cage which is longer in an anteroposterior direction than laterally. The sternum is likewise very prominent. None of the other bones seem to be affected and measurements of the limbs show them to be symmetrical.

Arising from the kyphosis and the angle between it and the ribs on either side hangs a large pedunculated tumor mass composed of loose tissue which is covered by small papil-

lomatous masses. On the right side this extends around the side until it almost reaches the midline of the abdomen. Another isolated tumor of similar type is found on the left thigh. Several other smaller lumps are found on various parts of the body. Over the main tumor the skin is a light brown color, and in other parts of the body are also seen discolored areas without tumor formation.

Roentgenograms show a kyphosis of the lower thoracic spine which is so marked as to form a "hair pin" turn (Fig. 1). The vertebrae at the apex of the deformity are fused into an irregular bony mass in which only traces of the outlines of the separate vertebral bodies can be seen. Examination of the remainder of the skeleton discloses no other abnormalities.

Biopsies were taken from the lump on the left thigh and the large mass in relation to the spinal deformity. Both showed the characteristic appearance of neurofibromatosis.

CASE II (Unit No. 352631). S. G., a white male, aged thirty-two, first noticed the appearance of pedunculated tumors in the skin at about eight years of age. These have greatly increased in size and number since that time. The patient's father has a similar skin condition. At about the time of the appearance of the skin lesions he bumped his right leg, which then gradually increased in size (Fig. 2). Ever since he can remember vision in the left eye has been poor. On physical examination a pulsating exophthalmos was found on the left with complete paralysis of the right rectus and impairment of the superior oblique and inferior oblique muscles. Considerable ptosis of the upper lid is also present.

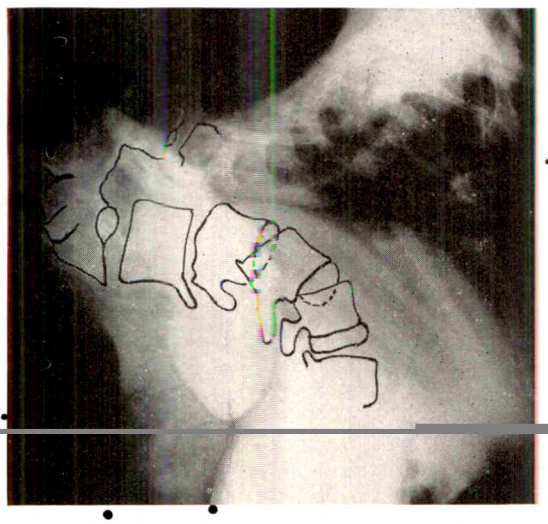


FIG. 1. Case I. *Neurofibromatosis with Congenital Deformity of Spine.* The deformity of the spine was noted at four and a half months of age. This has grown gradually worse, the maximum deformity being reached at seventeen years of age. A "hair-pin" kyphosis is present. The vertebral bodies at the apex of the deformity are fused into a solid bony mass, suggesting a congenital malformation.



FIG. 2. Case II. *Elephantiasis of Leg in Recklinghausen's Disease.* Patient first noticed pedunculated tumors in the skin at eight years of age. His right leg began to increase in size about the same time that the tumors in the skin appeared.

Roentgenograms of the skull disclosed a large defect in the lateral wall and roof of the left orbit with involvement of the anterior portion of the sphenoid bone on this side. Examination of the spine showed a rather marked scoliosis towards the right. A smoothly contoured concavity can be seen on the anterior aspect of the body of the fifth lumbar segment suggesting that erosion of the bone by a soft tissue tumor has taken place. Roentgenograms of the legs showed that the left leg is 3 cm. shorter than the right. The tibial shaft is widened in both diameters by the formation of irregular cortical



FIG. 3. Case II. *Neurofibromatosis of Tibial Shaft.* The tibial shaft is widened in both diameters by the formation of irregular, central and subperiosteal new bone. In the middle third of the shaft several smoothly contoured concavities can be seen at the margin of the bone caused by periosteal fibromas.

and subperiosteal new bone. In the middle third of the shaft several smoothly contoured concavities can be seen at the margin of the bone caused by periosteal neurofibromas (Fig. 3).

Biopsy of one of the skin nodules revealed the characteristic changes of neurofibromatosis.

The skeletal changes in this case agree in practically every detail with the bone changes of Recklinghausen's disease as described by Lehman and Brooks.[15] The large defect in the wall of the left orbit is in all probability due to bone erosion by a neurofibroma.

CASE III (Unit No. 556522). D. B., a white girl, aged twelve, had complained of a "swelling" of two or three years' duration at the site of an unerupted first molar in the lower right mandible. This has increased slowly in size until on admission it presents itself as a smooth tumor extending from the lower right third molar area anteriorly to the first bicuspid region. Roentgenograms (Fig. 4) show that the lower right first permanent molar is retained within the body of the mandible. Surrounding the tooth is a wide area of rarefaction which extends to the gingival margin. The margins of the rarefied area are well defined and show an increase in bone density suggesting that the defect in the bone was caused by a cyst. The alveolar margin of the mandible appeared to be destroyed. Another smaller area of diminished density surrounding a retained deciduous root or another tooth bud can be seen a little anteriorly.

Exploration of the rarefied area in the mandible revealed that it was filled with a white, firm soft tissue which seemed to merge and blend into the surrounding tissues. However, microscopically the margin of the mass was found to be well demarcated and composed of neurofibromatous tissue. With Cajal's silver stain scattered nerve processes could be seen in the tissue.

No other neurofibromata were found. The tumor probably arose on one of the nerves of the periodontal membrane which is richly supplied with nervous tissue and which corresponds to the periosteum in the long bones. No evidence was obtained that would indicate that the neurofibromatous tissue was infiltrating the

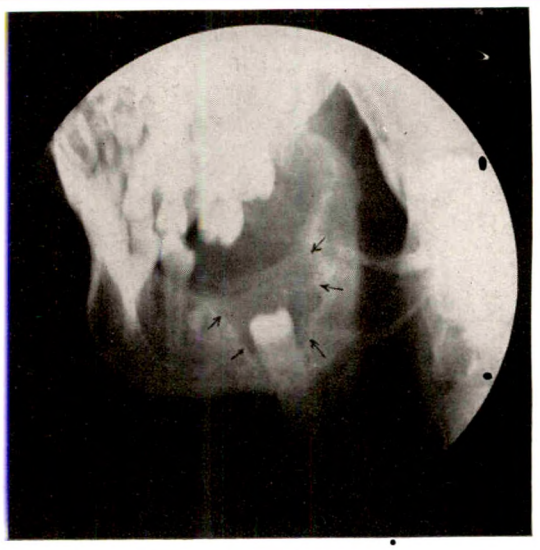


FIG. 4. Case III. *Neurofibromatosis of Mandible.* The patient complained of a swelling of two to three years' duration in the region of an unerupted first molar in the right lower mandible. The tooth is surrounded by an area of rarefaction with well defined margins which are increased in density. The alveolar margin has been destroyed.

bone. This case represents a localized form of neurofibromatosis.

CASE IV (Unit No. 288186). A. M., a white male, aged thirty-one, farm hand, first came under observation February 13, 1931. The chief complaint was progressive weakness of the legs of eight years' duration and diarrhea following meals of four years' duration. The weakness of the legs began first with marked tenderness of the feet and attacks of cramp-like pains in the legs at night. Later a spastic weakness of the legs developed which became so severe that he was able to walk only with great difficulty. The diarrhea, which began four years prior to his hospital admission, was explosive in character, coming on soon after meals. The stools were watery and frothy and accompanied by the passage of considerable gas. There was no nausea or vomiting. Relief was obtained by the use of opium and belladonna.

For as long as he could remember, the patient had a large patch of "warty" skin on the upper half of the body on the left side. It extended from the level of the left ear down over the shoulder anteriorly and posteriorly including the axillary region. The skin of the medial aspect of the face, arm and fingers was also involved. Several years ago the skin behind the left ear began to thicken and a mass appeared which has grown progressively larger. It now presents itself as a soft, deeply fissured, egg-shaped tumor which exudes an offensive watery discharge.

Physical examination disclosed an undernourished and poorly developed young man.



FIG. 5. Case IV. *Neurofibromatosis of Pelvis.* There is a unilateral involvement of the left half of the pelvis and bones of the lower extremity by an osteosclerotic process. The left innominate bone is smaller than the right. Several radiolucent areas can be seen in the region of the acetabulum suggestive of cyst formation. The upper extremity of the left femur showed linear striations of increased density.



FIG. 6. Case IV. *Neurofibromatosis of Femur.* The lower end of the femur is widened in both diameters. The cortex of the shaft is thickened most marked on its medial aspect. Several irregular cyst-like areas can be seen at the lower end of the shaft.

His head was unusually small. A definite asymmetry of the face, body and limbs was seen, the left side being smaller than the right. Because of the relative shortness of the left leg, the patient leans toward this side when standing. The left pupil was large, irregular and reacted sluggishly to light. The tongue was large, red, beefy and smooth on its edges. Both lower extremities were spastic and there was a bilateral Babinski. The deep reflexes of the legs were overactive. There was a transient ankle and patellar clonus. Vibratory sensation of the lower extremities was diminished to the iliac crests.

Laboratory studies, which included routine



FIG. 7. Case IV. *Neurofibromatosis of Tibia.* The cortex of the tibia is thickened, most marked on its anterior aspect. The margin of the bone is finely serrated. Coarse linear striations can be seen at the upper end of the shaft.

blood counts, serum phosphatase, proteins, calcium, sugar tolerance determinations, stool guaiac and gastric expressions, showed no remarkable variation from the normal.

Roentgenograms showed a unilateral involvement of the left half of the pelvis and bones of the lower extremity by an osteosclerotic process. The left innominate bone was distinctly smaller than the right. A number of relatively radiolucent areas were seen in the region of the acetabulum suggestive of cyst formation. The upper extremity of the femur showed broad linear striations of increased density (Fig. 5). The lower end of the femur was widened in both diameters, giving it a club-shaped appearance (Fig. 6). There was also bowing of the lower half of the shaft with the convexity directed anteriorly and slightly outwards. The cortex of the bone was thickened most markedly on its medial aspect while anteriorly the cortex was found to be thinner than usual. Several irregular, cyst-like areas were seen in the bone at the lower end of the shaft. The tibia also showed a thickening of its cortex similar to that seen in the femur (Fig. 7). This is most marked on its anterior aspect. Here the margin of the bone was finely serrated. Coarse linear striations can be seen at the upper end of the tibial shaft but there was no evidence of cyst formation such as seen in the femur and in the region of the acetabulum. In the bones of the left foot, the cancellous portion of the tarsals and the first metatarsal show irregular striations of increased density and some widening of the shaft of the first metatarsal.

Biopsies were obtained from the pectoral muscle and several small nodules were removed from both arms. These showed the characteristic changes of neurofibromatosis. Because of the unusual sclerotic changes manifested in the bone, a biopsy was also obtained from the lower end of the femur. The cortex of the bone was found to be covered with smooth periosteal tissue and showed no irregularities. A piece of bone measuring approximately 2 by $\frac{3}{8}$ inch in diameter was removed, exposing the marrow cavity. The cortex of the bone was found to be denser than normal and in the process of cutting the bone with an osteotome, several chips came away in layers suggesting an abnormal brittleness. Some of the trabeculae of the medullary cavity also seemed to be denser than usually encountered.

Microscopic examination of the cortical bone showed no recognizable changes. Tissue from the marrow, however, showed a growth in the marrow spaces which very closely simulates neurofibromatous structure (Fig. 8). Accom-

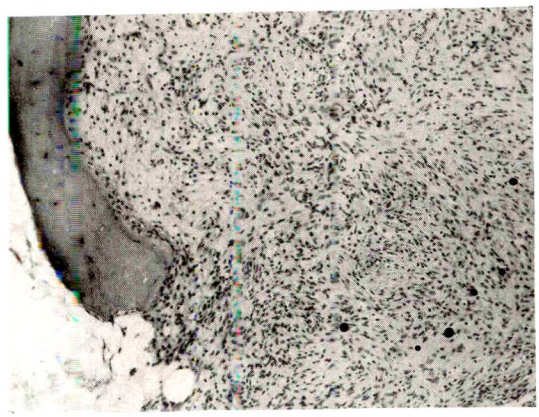


FIG. 8. Case IV. *Neurofibromatous Tissue in Bone Marrow.* Microsection from the marrow obtained from the lower end of the femur. The marrow spaces are filled with a neurofibromatous type of tissue.

panying this there are a great many lipoid containing foam cells and occasional small multinucleated giant cells. The presence of neurofibromatous type of tissue suggests that the bone changes were probably occasioned by direct involvement of the marrow space by the same process which has affected the peripheral nerves.

COMMENT

The presence of nerve tissue in both compact and cancellous bone has been amply demonstrated in man. Kölliker[12] in 1854 described in considerable detail the nerve supply of the bones. He listed Lushka, Kobelt, Engel and Beck among the earlier histologists who also had shown a nerve supply to the bones. Both sympathetic and cerebrospinal nerves have been found in the periosteum and it is from here that the chief nerve supply to the bones is derived. Minute trunks have also been described accompanying the nutrient vessels. The double source of nerve supply has been confirmed more recently by Stöhr.[25] He found a rich supply of nerves in the periosteum. From the periosteal nerves minute fibers enter the underlying bone through Volkmann's canals. Although nerve endings have been described by Maximow and Bloom[16] in extra-osseous connective tissue, they have never been identified in similar tissue within bone marrow. In connective tissue the nerve fiber first loses its myelin and then breaks up into fine terminal threads accompanied only by Schwann's nuclei. It does not seem unreasonable to assume, therefore, that the nerves in the bones terminate in the same way. Small nerves can usually be identified accompanying the larger blood vessels. Rarely an independent nerve trunk, largely autonomic in origin, has been observed. Nerve endings in the medullary tissues from this source have not been definitely identified.

In support of the presence of nervous elements in the marrow is the report by Peers[20] of a perineural fibroblastoma in this structure. De Santo and Burgess[5] also reported the occurrence of neurilemmoma in bone.

The relationship of the growth of neurofibromatous tissue in the marrow cavity and the sclerotic changes in the bone is not clear. Hyperostosis of the ilium was noted in one of the cases reported by Lehman and Brooks.[15] Weber[28] reported the case of a woman, aged forty-seven, with Recklinghausen's disease in which the shaft of the left tibia was greatly thickened and curved. Surrounding and completely covering the tibial shaft was a diffuse mass of thickened neurofibromatous periosteum. There was no evidence that the neurofibromatous growth had actually penetrated into the cortex of the bone. He attributed the hyperplasia of the underlying bone to the increased vascularity associated with the thickened neurofibromatous periosteum. Miller[17] observed areas of cranial hyperostosis that were in relation to overlying neurofibromatous soft tissue but no actual invasion of bone by neurofibromatous tissue had taken place. In the absence of neuromas overlying the sclerotic bone which might have produced these changes by chronic irritation, it might be supposed that this effect was caused by the neurofibromatous tissue in the marrow cavity itself. The presence of thickened medullary trabeculae in the tissue examined supports this view.

SUMMARY

The relationship of neurofibromata to peripheral nerves has long been recognized and the osseous changes associated with the development of neurofibromata from the nerves of the periosteum have been described.

Four cases of neurofibromatosis with bone involvement are herewith presented. In one instance neurofibromatous tissue was demonstrated in the marrow cavity of the femur. This suggests that nervous tissue must be present in bone marrow.

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GAS IN THE FETAL CIRCULATORY SYSTEM AS A SIGN OF INTRAUTERINE FETAL DEATH

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THIS case is reported to record a sign of intrauterine fetal death which was demonstrated within sixty-six hours after the absence of fetal movements was noted. To the best of our knowledge this has not been reported previously.

CASE 1. Patient, aged twenty-two, para II, gravida III, a controlled diabetic, approximately at full term, noticed the absence of fetal movements on the afternoon of April 9, 1943, and was examined roentgenographically April 12 in the morning. Both previous pregnancies had been allowed to go to full term and normal babies of 8 and 9 pounds had been born. The patient had no untoward clinical symptoms.

Roentgen Examination. On April 12, 1943, sixty-six hours after the absence of fetal movements had been noted a roentgenogram in the anteroposterior projection (Fig. 1) was taken with the patient erect to show the fetal skull. The cranial bones show no overlapping and

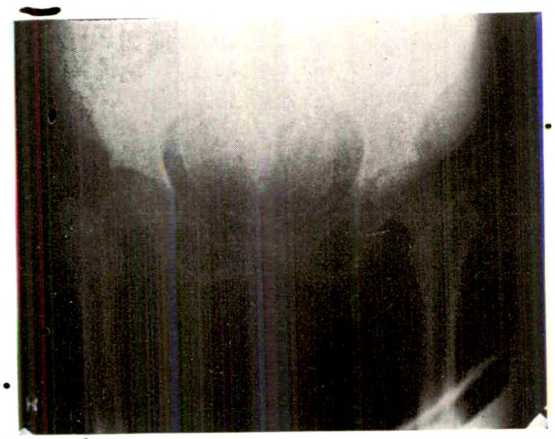


FIG. 1. Patient erect. Skull symmetrical.

give a symmetrical shadow. A roentgenogram in the right lateral projection with the patient horizontal (Fig. 2) and using a wedge-shaped filter as described by Vaughan[6] shows the shadow of a large, full term fetus with a protuberant abdomen and a well extended vertebral column.

The following areas of decreased density are noted: (1) A spiral of fairly constant diameter can be traced from the region of the fetal abdominal shadow to the peripheral zone of the uterine shadow. (2) Two parallel narrow lines

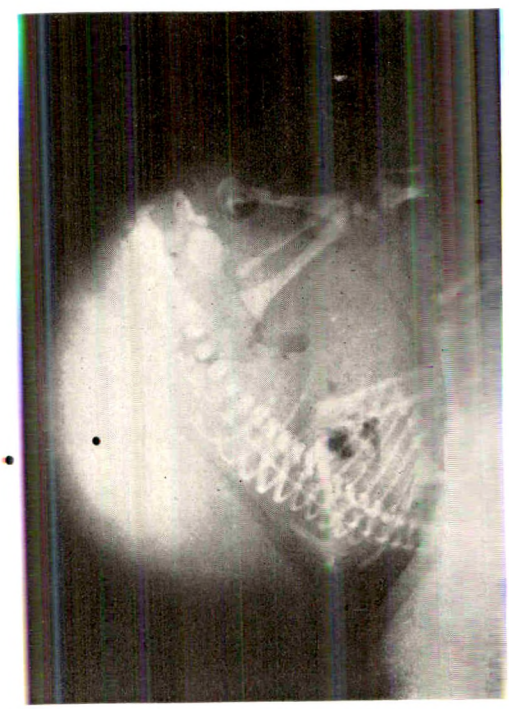


FIG. 2. Lateral projection, patient horizontal. Wedge-shaped filter of Vaughan[6] used.

traverse the shadow of the lower portion of the fetal abdomen, extending from the region of the umbilicus to the region of the pelvis. (3) A tree-like radiolucent area is present over the region of the fetal liver. (4) Over the fetal thoracic shadow a bilocular area is shown, which, due to its size, shape and position, suggests the demonstration of the cardiac chambers. (5) A large oval area is present over the shadows of the upper four fetal lumbar vertebral bodies.

The placenta is implanted on the anterior wall of the upper uterine segment.

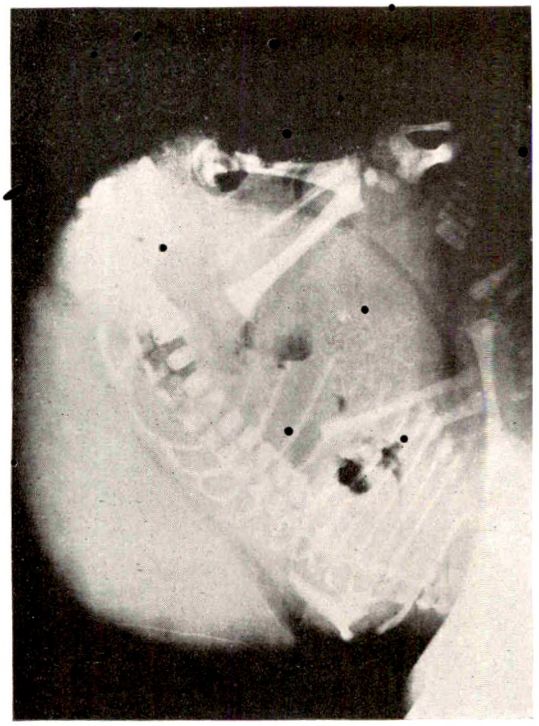


FIG. 3. Lateral projection, forty-eight hours after Figure 2. Slight increase in size of areas of decreased density.

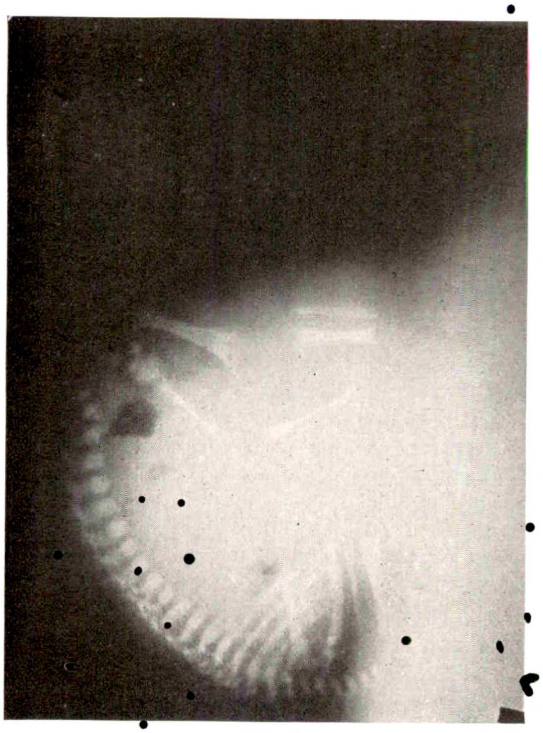


FIG. 4. Patient erect. Fluid levels showing

Conclusion: The areas of decreased density noted above are interpreted as evidence of gas in the fetal circulatory system. As such, they indicate that the fetus is dead.

April 14, 1943. The roentgenogram (Fig. 3) of the abdomen with a right lateral projection shows the radiolucent areas demonstrated on April 12, and these are increased very slightly in extent. The fetal extremities are in practically the same position as at the earlier examination and their shadows in these serial roentgenograms can be almost exactly superimposed.

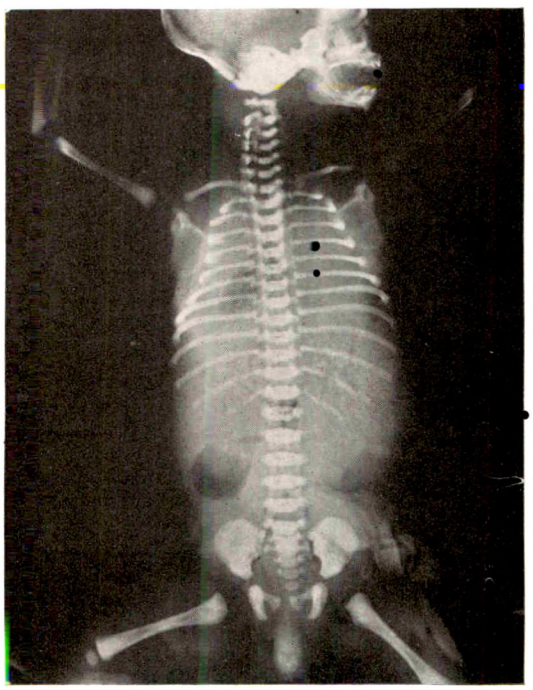


FIG. 5. Anteroposterior roentgenogram of stillborn baby immediately after birth.

A further roentgen study (Fig. 4) was made with the patient in the upright position and this shows fluid levels in at least two of the areas of decreased density, no abnormal flexion of the fetal skeleton and no overlapping of the fetal skull bones.

Conclusion: The earlier diagnosis of intrauterine fetal death is corroborated.

Delivery. Medical induction of labor was instituted and an uncomplicated delivery terminated April 15 at 11:00 A.M. The liquor amnii was not discolored and there was no abnormal odor.

Roentgen Examination of the Fetus and Placenta (Fig. 5 and 6). This examination was performed immediately and showed more clearly the areas of decreased densities noted above. These radiolucent areas appeared to demonstrate portions of the fetal circulatory system and the following structures were identified: both umbilical arteries and portions of the umbilical vein; the common iliac arteries; external iliac arteries; the femoral arteries; the abdominal aorta; blood vessels of the liver and mesentery; the chambers of the heart; pulmonary vessels; carotid, facial and intracranial vessels. The exact location of the radiolucent area in the left side of the abdomen was not determined.

Autopsy Report. Within an hour of delivery an autopsy was performed by Dr. G. A. McCurdy, Pathologist of the Royal Jubilee Hospital, Victoria B.C., and his report is herewith summarized.

The body was that of a full term male baby with a marked degree of maceration of the skin and desquamation. The cranial bones were partially separated and the brain was completely liquefied. The heart was opened under water and gas escaped from both right and left ventricles. The liver was opened under water and gas escaped from the cut surfaces. The tissues around the left kidney were infiltrated with gas. The umbilical cord was opened under water and gas escaped. All organs showed a marked degree of autolysis.

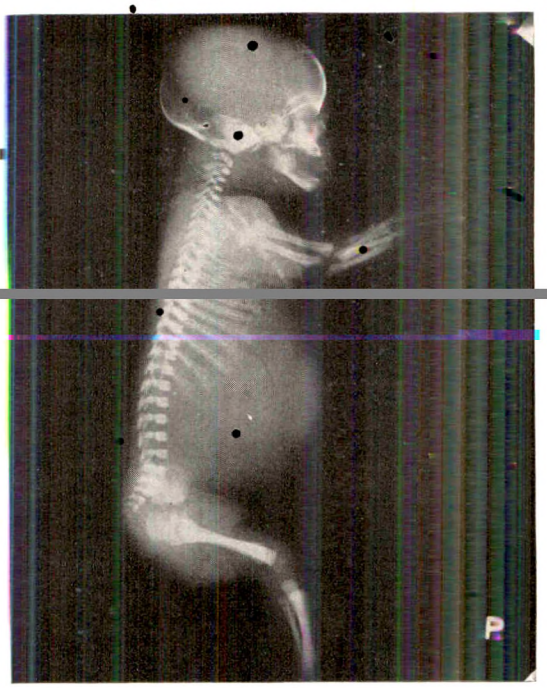


FIG. 7. Case II. Roentgenogram made immediately after birth of stillborn baby. Gas is present but not as clearly shown as in Case I.

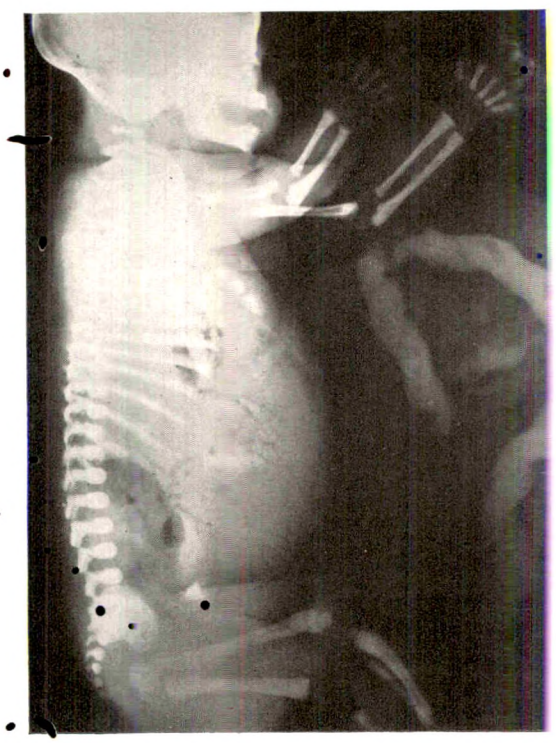


FIG. 6. Lateral roentgenogram taken at same time as Figure 5. Much of the circulatory system is clearly demonstrated.

Anatomical Diagnosis. (1) Marked maceration and autolysis of a stillborn baby; (2) presence of gas in heart, liver, retroperitoneal tissue and umbilical cord.

ACCEPTED SIGNS SUGGESTIVE OF INTRAUTERINE FETAL DEATH

1. Overriding of the skull bones with cephalic asymmetry recorded by Horner[2] and by Spalding.[3] Such a change has been demonstrated in a living fetus and reported by Chalmers.[1] The above case showed a symmetrical cranial shadow.

2. Marked bending of the spinal column, given by Szellö[4] as the earliest sign. In the

above instance the vertebral column was unusually extended.

3. Lack of change in the position of the fetal bones of the extremities in serial roentgenograms suggested by Thoms.[5] This immobility was very well demonstrated in our case.

4. Failure of the size of the fetal bones to correspond to the duration of pregnancy.

5. Haziness of landmarks and contours, or entire absence of bony shadows in repeated examinations.

CASE II. During the preparation of the above report examination of another full term, slightly macerated fetus was possible and this study is included (Fig. 7). This roentgenogram, taken with a lateral projection immediately after birth, shows areas of decreased density similar to those demonstrated in Case I, and these are interpreted as evidence of gas in the fetal circulatory system.

SUMMARY

1. A roentgenological diagnosis of intrauterine fetal death was made by the demonstration of gas in the fetal circulatory system.

2. A second case showing gas in the circulatory system of a stillborn fetus is reported.

3. Study of this phenomenon and of the nature of the gas is being continued and further reports will follow. It is suggested that careful examination with the lateral projection of patients suspected of intrauterine fetal death may show that this sign is of primary importance.

I am deeply indebted to Dr. H. H. Murphy, Director of the Radiological Department of the Royal Jubilee Hospital, Victoria, B. C., for this helpful direction and valuable advice; to Dr. G. A. McCurdy, Director of the Pathological Department; to Dr. G. B. B. Buffam, whose case I reported and to Dr. M. W. Calvert, of our Interne Staff, for his skillful preparation of the illustrative material.

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DISLOCATION OF THE HIP ASSOCIATED WITH SPINA BIFIDA*

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IN HOSPITALS and sanitaria devoted to the care of chronic diseases, there is an opportunity to observe the progress and end-results of chronic diseases not as a rule seen either in office practice or in hospitals caring for acute diseases. We have had occasion for this reason to observe 3 cases of spina bifida in conjunction with dislocation of the hip which we followed for prolonged periods of time until their death occurred from intercurrent complications. The association of dislocation of the hip with spina bifida is a recognized entity, yet very few instances are reported in the literature and we believe that a report of 3 additional cases should be of interest. The above findings occurred in 3 girls born with visible tumor formation in the lumbosacral region of the spine. Each was operated on soon after birth for the spina bifida. Two died at the age of twelve from an ascending urinary infection and the third died at the age of twenty from an unknown cause; this case also showed signs of urinary tract involvement.

REPORT OF CASES

CASE 1. E. R., female, aged fifteen, was admitted to the institution on July 23, 1936. She was a fourth child of six, was a full term baby, and had been delivered spontaneously. At birth a protruding mass was noted in the sacral area, and when the patient was five weeks old she was operated on. She remained at home until she was six years old and later on spent her life in various institutions. As a child her head seemed unduly large but diminished in size in later years. She was able to sit up at three years of age but never developed control of her sphincters. At an early age she underwent several orthopedic operative procedures to improve her gait, with some beneficial effect.

Physical examination at the time of admission disclosed an obese girl with fair mentality. She was of short stout stature and showed precocious physical development. She was able to walk on crutches. The muscle power survey revealed ability to flex the right hip in the recumbent position up to 60°, flexion of the knee to about 90°, with no dorsiflexion of the ankle. There was no motor power of the abductor muscles. On the left there was very little motor power of the leg. Passive flexion of the knee was possible to 45°. Genu valgum of the left side was noticed amounting to 25°, 15° of which represented a fixed deformity and 10° was due to laxity of the internal ligaments of the knee joint. In addition genu recurvatum was present. Marked lordosis with prominence of the sacral area was evident. Left foot was in varus position. Sensation was absent from the lower legs down. Over the sacrum and over both buttocks there were numerous small decubitus ulcers. The deep reflexes were diminished bilaterally, weaker on the left side, and the Achilles tendon reflex on the left side was absent.

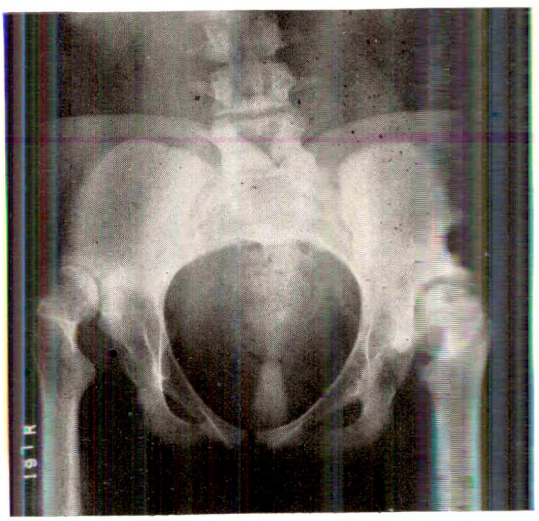


FIG. 1. Case 1. Extensive defects of the posterior segments of the fifth lumbar vertebra and sacrum. Bilateral dislocation of the hips with false acetabular activities, and evidence of a shelf operation on the left side.

* From the Radiological Service of the Jewish Sanitarium and Hospital for Chronic Diseases, Brooklyn, New York.

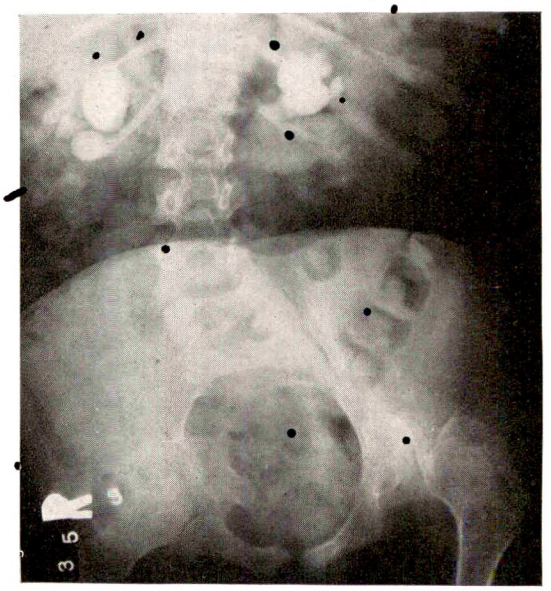


FIG. 2. Case II. Defects of posterior segments of the third, fourth and fifth lumbar vertebrae and the entire sacrum. Dislocation of the left hip. Bilateral hydronephrosis.

Laboratory examination showed the presence of albumin and white blood cells in the urine on several occasions.

She died at the age of twenty with no apparent immediate cause of death discernible except for evidence of an ascending urinary infection. She was found dead in bed after being up in the ward with other patients the night before.

Roentgen studies revealed almost complete absence of the posterior segments of the fifth lumbar and first, second and third sacral segments. There was a bilateral dislocation of the hips and formation of false acetabular cavities on both sides. The head and neck of the femur on the right appeared normal. On the left the head and neck showed considerable distortion and there was also evidence of a previous surgical procedure with the formation of a shelf on the left side (Fig. 1).

CASE II. G. G., female, was admitted to the hospital on December 10, 1936, at the age of seven. She was the first child of her parents and was delivered in breech presentation after three days of labor. A visible tumefaction of the lumbosacral area was removed by operation when she was one week old. She was never able to control the sphincters. Until three months old she did not move her legs at all; however, later she gradually developed the ability for gross movements of the legs but was never able to move her toes and never learned how to walk. She stayed at home until three years of age and later was hospitalized in various institutions.

Physical examination on admission disclosed an alert child with a tendency towards obesity. She stood with a backward displacement of the pelvis and an exaggerated lumbar lordosis. Motor power survey revealed the gluteal muscles on both sides to be markedly atrophied. The power of the right quadriceps was fair. The abductors, adductors and rotators were fair. The hamstrings were weak. There was no motor power in the leg and foot muscles. On the left side the thigh muscles were fair except the abductors and external rotators. Both knees showed a genu recurvatum deformity. The deep reflexes were absent bilaterally; analgesia below the fourth lumbar vertebra was found, the position sense was lost, and the vibratory sense was absent in the toes but somewhat perceptible in the tibia and ankle. Bilateral foot drop was present.

Three years after admission the patient began to have spisodes of spiking temperatures. Urinalysis showed the presence of albumin, red blood cells, and white blood cells. The patient would improve after therapy for a short period and the symptoms would recur soon afterwards.

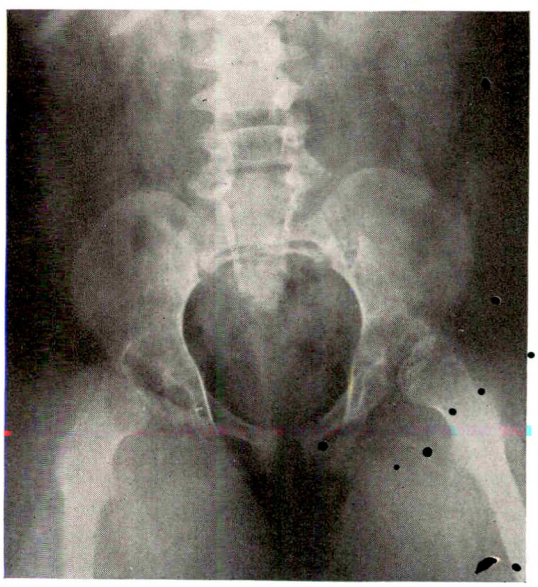


FIG. 3. Case II. The spinal defect is clearly shown. The left hip is now in relatively normal position and easily replaced.

The clinical course was later characterized by marked anemia and urea nitrogen retention. She died as a result of a pyelonephritis at the age of twelve.

Roentgen examination showed the presence of an extensive defect involving the posterior segments of the third, fourth and fifth lumbar vertebrae and the entire sacrum. The left hip was dislocated. The left femoral head appeared relatively well developed. There was a slight flattening of the capital epiphysis. The left hip could be easily replaced but would just as readily dislocate itself. The pyelogram showed marked bilateral hydronephrosis.

This case illustrates the rôle of the muscle imbalance in the production of the dislocation. (See Figures 2 and 3, before and after dislocation.)

CASE III. R. P., female, was admitted to the hospital at the age of twelve. She was an only child. The family history was essentially negative. When she was two days old a spina bifida of the lumbosacral area was surgically removed. She never developed control over the sphincters and never was able to walk without assistance. At the age of two she developed pyelitis. The infection was severe and only after repeated transfusions did she recover. Frequent ureteral catherization was necessary subsequently to relieve exacerbations of urinary symptoms.

Physical examination on admission revealed a rather thin girl of excellent mentality. She complained of constant pain in both lumbar areas. Muscle survey showed fair power in both quadriceps muscles and internal rotators. The abductors and external rotators were paretic. There was no motor power in the leg and ankle muscles. There was bilateral drop foot. The knee and ankle reflexes were not elicited, and there was complete loss of sensation below the fifth lumbar vertebra.

Laboratory examination disclosed the presence of albumin, white blood cells, and red blood cells in the urine. Examination of the blood revealed the presence of a marked anemia.

This patient died soon after admission of uremia at the age of twelve.

The roentgenograms showed the presence of a defect involving the posterior segments of the fourth and fifth lumbar and the entire sacral spine, also marked distortion of the lower end of the sacrum and coccyx. Bilateral dislocation of the hips was present with upward riding of both femora (Fig. 4). The development of both femora including the head and neck on both sides aside from generalized atrophy was essentially negative. The acetabula, however, were not well developed. An intravenous pyelogram showed a bilateral hydronephrosis.

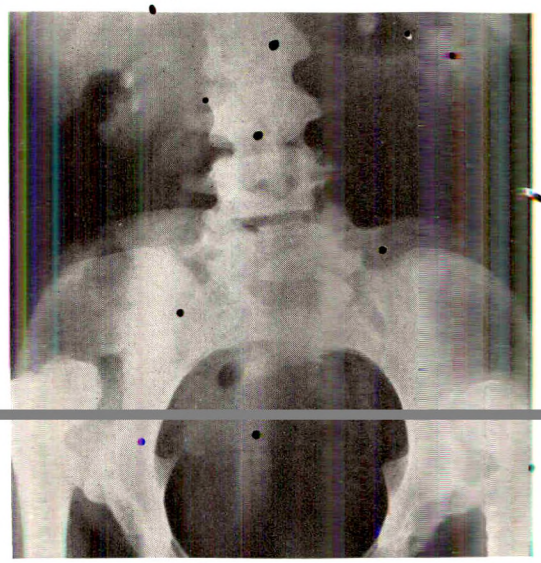


FIG. 4. Case III. Irregular defect involving the posterior segments of the fourth and fifth lumbar vertebrae and the entire sacral region. There is a bilateral dislocation of the hips with upward riding of both femora. There is evidence of a bilateral hydronephrosis shown by an intravenous pyelogram.

DISCUSSION

Spina bifida is a congenital developmental defect of the neural canal characterized by imperfect closure of the spinal lamina. Associated with this defect are abnormalities of the overlying skin and subcutaneous tissues and of the underlying meninges and spinal cord. It is most common in the lumbar and sacral regions but may be encountered in the cervical and thoracic regions. In its severe forms it is accompanied by other anomalies, the most important of which are: anencephaly, hydrocephalus, talipes equinus, harelip, cleft palate, and so forth. Myelocele, meningomyelocele, meningocele, and spina bifida occulta are only differences in degree of severity of involvement.

Clinical Features. When the protruding sac is exposed and susceptible to infection, these patients seldom survive for very long without surgical correction. In the milder cases, owing to the survival of the patients, time is allowed for the development of the more remote effects of the neural involvement. As the abnormality most commonly occurs in the lumbosacral region, the neurological signs manifest themselves in the lower limbs. Frequently one group of muscles show fair power whereas antagonistic muscles may show complete paralysis due to the neural defect. This combination of paralysis in one group of muscles and fair motor power in another group was present in our cases. Sphincter disturbances are common, the most important being incontinence of urine. Fecal incontinence occurs but is less frequent.

In the pathogenesis of dislocation of the hip two types are distinguished: the endogenous type, the result of developmental arrest in the acetabular roof in contrast to the dynamic type caused by the presence of abnormal forces which are responsible for the dislocation of the femoral head from the acetabulum. In the cases reported the dislocation suggests a paralytic origin brought about by two factors: the relaxation of the ligaments around the hip, and the muscle imbalance. The dislocation usually occurs in the absence of muscle power within the abductors and external rotators. In our 3 cases this muscle imbalance was demonstrated by the muscle survey. The maldevelopment of the acetabular cavities and the atrophy of the femora may be accounted for by the factor of disuse rather than by endogenous involvement of the acetabulum, which produces congenital dislocation.

SUMMARY

Three cases are presented showing the association of spina bifida and dislocation of the hips. From the physical and roentgen examination we believe that the dislocations are the result of the neural defect produced by the spina bifida and not the result of multiple anlage defects, although the latter cannot be ruled out. These cases are of interest because of the relative infrequency of this complication of spina bifida and the few cases reported in the literature.

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WERNER'S SYNDROME

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IN 1934 Oppenheimer and Kugel[5]* reported for the first time in this country on a heredofamilial disorder consisting of scleroderma, bilateral juvenile cataracts, precocious graying of the hair, and other endocrine stigmata which they called Werner's syndrome. In the European literature 10 similar cases have been reported. These include papers by Werner,[8] Bloch and Stauffer,[2] Rothmund,[7] Barbot,[1] Krebs, Hartmann and Thiébaut,[3] and Mamou.[4] There are only 3 cases in the American literature, all of them from Montefiore Hospital where they were studied for many years. All 3 patients are now dead and have been autopsied. They showed remarkable similarities in their clinical and roentgenologic appearances. It is the purpose of this paper to describe the roentgenographic findings in the hope that more cases of this unusual and interesting syndrome will be added to the 3 already described.

The etiology of the disease is obscure. However, there is no doubt that the disease is familial. This is definitely shown in the cases published by Werner and by Barbot. Two of the 3 patients to be discussed in this paper were brothers. Although the etiology is obscure, the disease is essentially a degenerative one, probably caused by some defect in the mesodermal and ectodermal tissues.

The clinical syndrome is characterized mainly by premature senescence associated with some features of juvenilism. There is generalized underdevelopment of all the endocrine glands resulting in hypogenitalism, gynecomastia and eunuchoidism. In the female there are menstrual disorders, including premature total amenorrhea occurring as early as sixteen years of age. Occasionally some evidence of thyroid insufficiency may be present.

Vascular changes are an outstanding feature of the disease. There is clinical evidence of extensive and widespread arteriosclerosis of the Mönckeberg type beginning in the third decade of life. The peripheral vessels are tortuous and non-compressible. The major clinical changes, however, appear in the ectodermal structures. Whether these are secondary to vascular impairment or to changes primarily in these structures is difficult to evaluate.

Skin. The predominant changes in the skin suggest a combination of scleroderma and poikiloderma. (The precise status of these changes is still a matter of dispute.) This is most marked in the distal portions of the extremities, especially in the lower extremities where it is often associated with recurrent ulceration and subsequent gangrene necessitating amputation.

Hair. All cases show premature graying of the hair, with onset as early as the eighth year. The graying is rapidly followed by alopecia.

Cataracts. Bilateral cortical cataracts are a constant finding. These generally begin in the third decade of life and progress rapidly, so that early in life these patients need surgical intervention to prevent blindness.

Voice. The voice is changing in character and has been described as rasping and high pitched.

Pathological examination of 3 patients has contributed very little to our understanding of the etiology or pathogenesis of this disease. All 3 patients showed a striking resemblance during life. They were often mistaken for one another. All died at approximately the same age—forty-one, forty-two and forty-four years. It is interesting that the cause of death was different in each; one died of myocardial failure, as

* The cases described by these authors are the ones used in this paper for the evaluation of the roentgenographic findings.

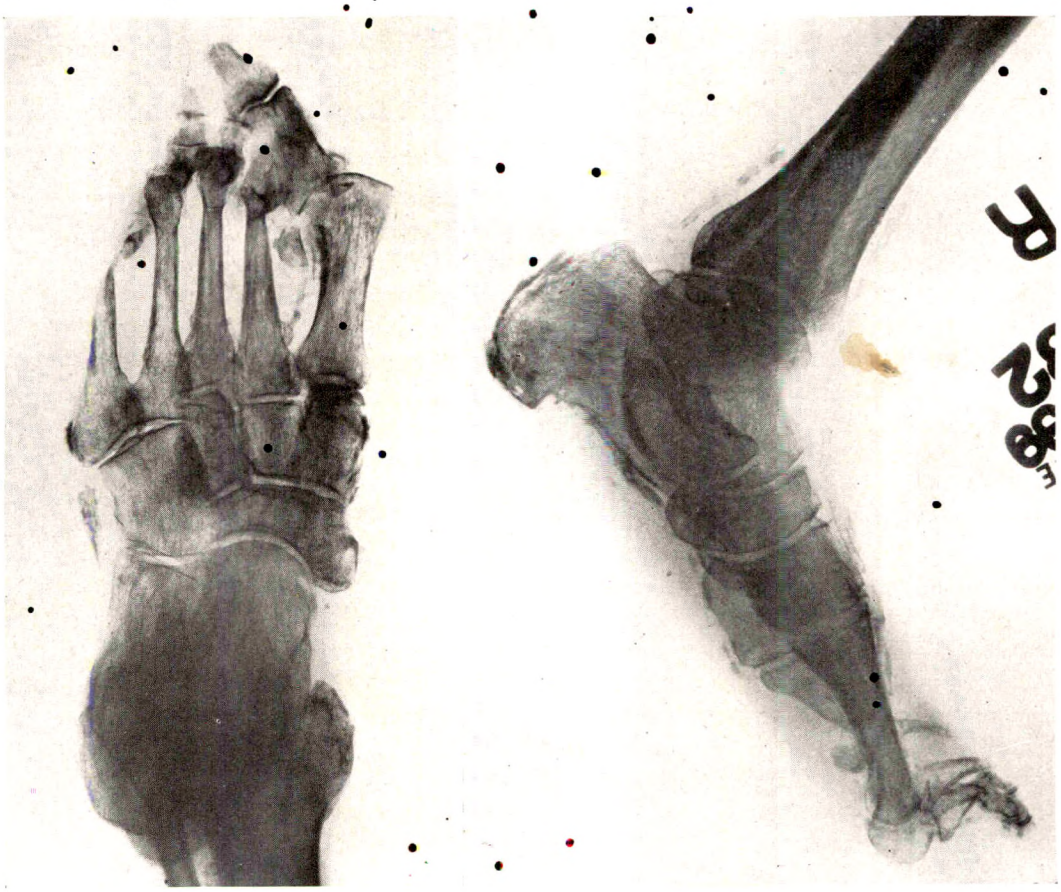


FIG. 1

a result of far advanced coronary disease; another died of a primary carcinoma of the liver with subsequent metastasis, and the third died of a fibrosarcoma of the forearm with extensive pulmonary metastasis.

One of the 3 cases showed evidence of increased parathyroid activity. This was evidenced microscopically by the increased numbers of large clear cells. During life, this patient showed some findings suggestive of hyperparathyroidism. These included a positive Hamilton test and calcium levels up to 13.0 mg. However, the other 2 cases had small parathyroid glands and no clinical evidence of hyperactivity. The blood phosphatase in all cases ranged from 4 to 8 Bodansky units.[9] The other endocrine glands were small and atrophic.

ROENTGENOGRAPHIC FINDINGS

The roentgenographic findings are characterized by extensive calcification in the arteries and soft tissues.

(1) *Arterial Calcification.* There is extensive calcification of the Mönckeberg type. This involves all arteries except those of the brain and lungs. The changes are most marked in the distal parts of the extremities (Fig. 1 and 2), especially the lower extremities, where one can identify all the arteries including the plantar and dorsal arches, as well as their digital branches. The same is true of the hands, but to a lesser degree. Extensive calcification is also seen in the abdominal vessels, particularly in the mesenterics (Fig. 3).

(2) *Calcification in the Soft Tissues.* Calcium was usually deposited in ligaments, tendons, bursae and subcutaneous tissues. At times it is difficult to determine in which of these structures the calcium is located, since they are so often intimately related.

All 3 cases showed calcification in or about the plantar calcaneocuboid ligaments, the metatarsophalangeal joints, both malleoli and the calcaneus at the point of attachment of the Achilles tendon (Fig. 1).

The knee joint also showed widespread involvement. There was calcification of the medial and lateral collateral ligaments, as well as the patellar ligament, and quadriceps femoris tendon (Fig. 4). Calcification was less marked about the hip joint where only 1 of the 3 cases showed some calcification. This was in the region of the trochanteric bursa. Calcification in the upper extremities was present in the region of the olecranon bursa, and the interphalangeal joints (Fig. 2 and 5). None of the 3 cases showed calcification of the subdeltoid or subacromial bursa. Two of the 3 cases showed small calcific deposits varying from pinhead to pea size scattered through the subcutaneous tissues over the tibia. These also were seen in the soft tissues about the small joints of the hand (Fig. 2).

(3) *Skull.* Examination of the skull revealed calcification of the pineal and an unusually small sella turcica in all cases (Fig. 6).

(4) *Larynx.* Roentgenoscopic examination of the larynx revealed rigidity of the epiglottis during the act of swallowing.*

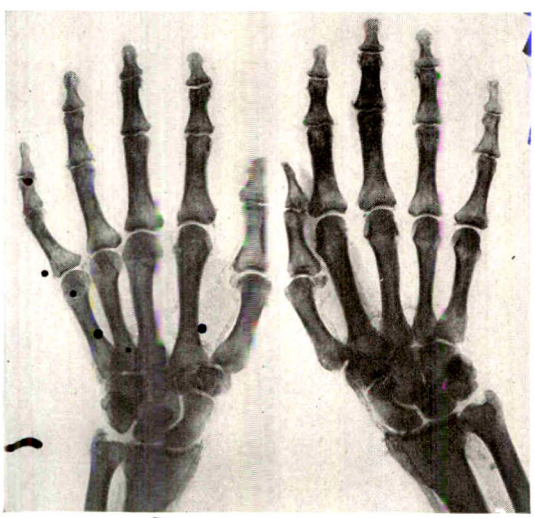


Fig. 2

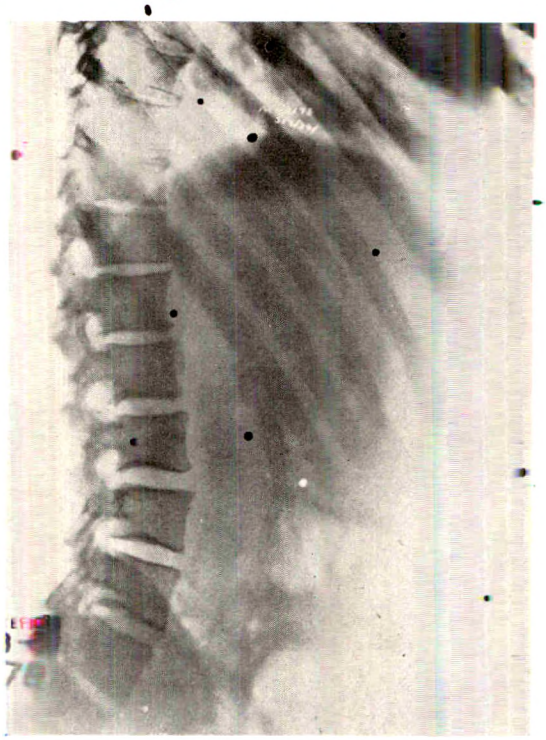


Fig. 3

(5) *Osteoporosis.* Osteoporosis was confined mainly to the lower extremities. It was not extensive and could easily be explained on the basis of local disuse rather than any generalized disorder.

(5) *Periostitis.* One of the 3 cases showed periostitis over the tibia. This most likely was secondary to chronic ulceration and infection of the overlying skin and subcutaneous tissues.

Differential Diagnosis (Roentgenographic)

(a) *Increased Parathyroid Activity* (primary or secondary). In increased parathyroid activity one not uncommonly sees metastatic calcification of blood vessels and soft tissues. However, in Werner's syndrome the calcification is entirely out of proportion to the osteoporosis. Osteoporosis is localized to the extremities. It is not severe and does not show cystic changes.

* Personal communication from Dr. Selma Shapiro.

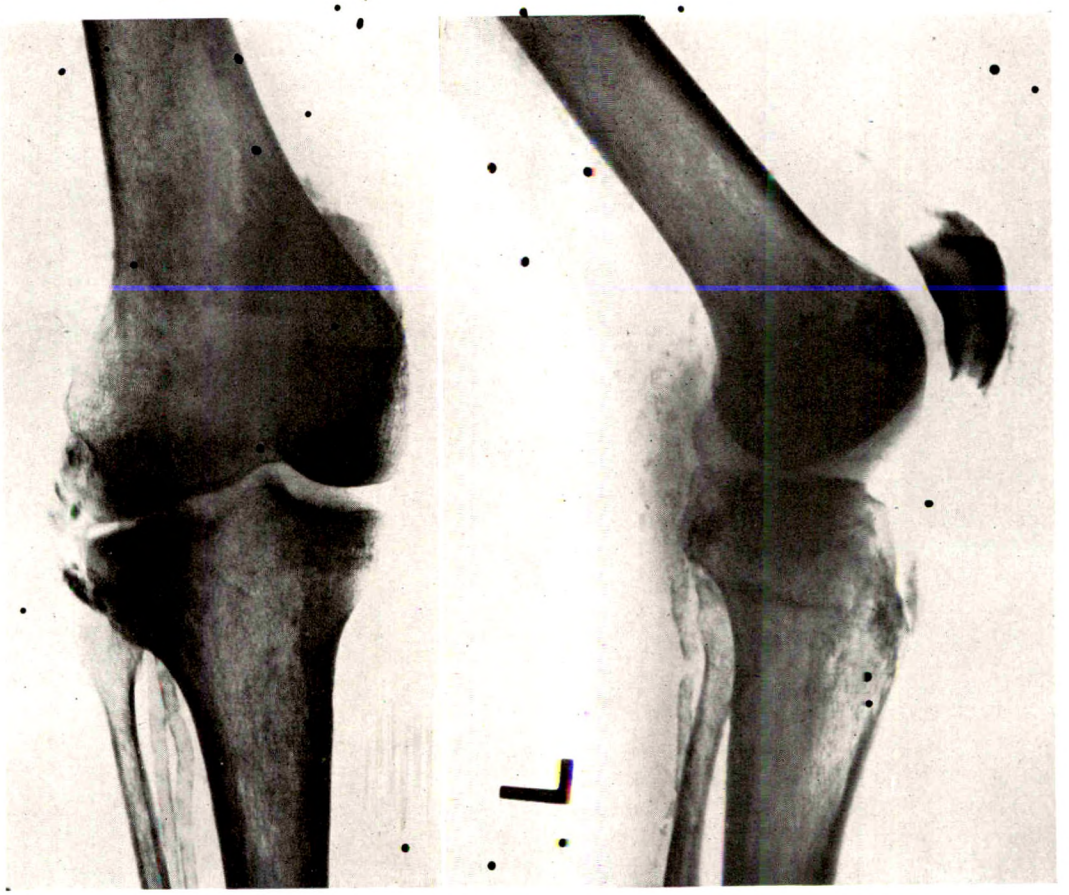


FIG. 4



FIG. 5

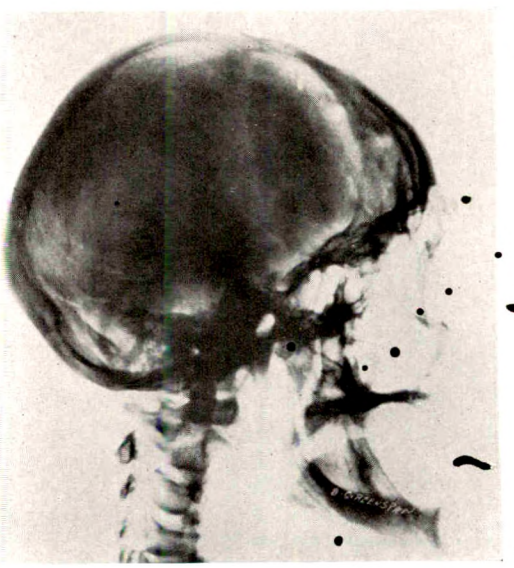


FIG. 6

(*b*) *Arteriosclerosis* (Mönckeberg). In advanced arteriosclerosis calcifications are not seen in the soft tissues. The relationship of the extent of arterial calcification to the age of the patient is of great importance in differentiating the two diseases.

(*c*) *Scleroderma*. In scleroderma calcifications are occasionally found in the subcutaneous tissues. However, there is no associated calcification of arteries, bursae, or ligaments. Scleroderma may result in absorption of the terminal tufts of the fingers and toes which is not seen in Werner's syndrome.

Where the differential roentgen diagnosis becomes difficult or impossible the clinical findings are generally sufficient to establish the diagnosis of Werner's syndrome.

SUMMARY

The roentgenographic findings in three cases of Werner's syndrome are presented. These include:

1. Metastatic calcification in the soft tissues, tendons, ligaments and bursae.
2. Extensive calcification of the media of all arteries except those of the brain and lungs.

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MAY, 1944

THE AMERICAN JOURNAL OF ROENTGENOLOGY AND RADIUM THERAPY

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Twenty-eighth Annual Meeting: 1944, to be announced.

EDITORIALS

THE ROENTGENOLOGICAL APPEARANCE OF THE GASTROINTESTINAL TRACT IN SCLERODERMA

THE clinical and microscopic aspects of generalized scleroderma have been well studied. Most of the textbooks dealing with diseases in which scleroderma is discussed confine their discussion to the skin manifestations and the histopathology of the skin lesions, neglecting to mention the fact that esophageal dysfunction may occur early in the disease before it has assumed its characteristic picture. However, this feature of scleroderma was reported as early as 1903 by Ehrmann and several later reports have occurred in the literature, one of the most recent reports dealing with lesions of the esophagus in generalized progressive scleroderma being by Lindsay, Templeton and Rothman.[1]

From a study of their cases they too observed that esophageal disturbance is a characteristic manifestation of generalized scleroderma and that it is likely to appear early in the course of the disease. These esophageal disturbances are moreover in the early stages attributed to gastric dysfunction or they may be of such mild nature as to receive little or no attention. These early esophageal symptoms manifest themselves in inability to swallow more than a few mouthfuls of fluid rapidly, difficulty in swallowing while lying down and a sensation of fullness behind the sternum.

During their roentgenoscopic studies Lindsay, Templeton and Rothman observed that normal peristalsis is absent in the lower two-thirds of the esophagus and that the contents accumulate in this relaxed and dilated portion. The burning sensation in the chest in the region of the epigastrium, and neck, of which many of these patients complain, suggests the probability of regurgitation of gastric contents into the esophagus and resulting chronic esophagitis. The roentgenoscopic observations also brought forth evidence that this suggestion was well founded in fact in that the cardiac sphincter as well as the cardiac musculature above was affected by some degree of weakness.

It was also noted that chronic ulceration in the lower third of the esophagus localized chiefly to the region just above the phrenic ampulla. This chronic ulceration they attributed probably to a direct result of the esophagitis, with sclerodermatic changes as a predisposing factor.

In the later stages of the disease stricture formations of the esophagus occurred, limited to the region immediately above the phrenic ampulla.

While the esophageal disturbances associated with scleroderma have attracted the attention of many observers, the roentgenological appearance of the gastrointestinal tract in scleroderma, particularly the changes seen in the small and large intestine, have not been discussed until recently, except in the report of an isolated case by Rake.

Hale and Schatzki,[2] in the April issue of this JOURNAL, reported studies which they have made during the past five years at the Massachusetts General Hospital on twenty-two patients who were known to have scleroderma varying in duration from five months to twenty-two years. The roentgen examination in seven of this group of patients was carried out because of difficulty of swallowing of which the patients com-

[1] Lindsay, J. R., Templeton, F. E., and Rothman, S. Lesions of the esophagus in generalized progressive scleroderma. *J. Am. M. Ass.*, 1943, *123*, 745–750.

[2] Hale, C. H., and Schatzki, R. The roentgenological appearance of the gastrointestinal tract in scleroderma. AM. J. ROENTGENOL. & RAD. THERAPY, April, 1944, *51*, 407–420.

plained. However, the fifteen remaining cases had no symptoms referable to the esophagus and the examination in this group was carried out routinely for the purpose of determining whether or not there were changes in the stomach or intestinal tract somewhat comparable to or simulating those which had been observed in the esophagus.

Of the eighteen patients who had scleroderma who were examined roentgenologically only four showed changes in the small intestine. These changes that were noted were quite apparent and appeared to be of considerable clinical interest and importance as all of these four cases had symptoms referable to the gastrointestinal tract which indicated partial obstruction. The findings in the four cases that exhibited positive roentgen changes in the small bowel pattern were characterized by widening of the part of the small intestine involved, with marked delay in the emptying time of these loops. The dilatation involved the upper small intestine including the duodenum in all four patients and extended downward into the ileum in one, and in this case where the lesion was so extensive there appeared to be involvement of the colon as well.

These roentgen changes observed in the small intestinal loops must be differentiated from so-called deficiency diseases, in which the small bowel pattern has been so aptly described by Golden, and these changes must not be confused with paralytic ileus. The differentiation from paralytic ileus is fairly easy since the pathologic changes usually involve only a certain part of the intestine and the clinical symptoms of the patient are never so acute in scleroderma as those found in paralytic ileus. However, the similarity of the changes seen in severe cases of deficiency disease to those in scleroderma is somewhat striking, the difference, however, being that the abnormalities seen in the small intestine in scleroderma appear to be more localized. Clinically apparently none of the patients exhibiting scleroderma showed evidence of avitaminosis and those patients having scleroderma who were treated intensively with vitamins showed no improvement in the appearance of the small bowel pattern.

While Hale and Schatzki could not prove that the changes seen in scleroderma were not produced by some deficiency, it was their impression and belief that the changes in the small intestine have some similarity to those in the esophagus, and while it was not the purpose of their paper to discuss the origin of the changes observed in the gastrointestinal tract they thought that these might be explained on the basis of local intestinal changes or of some neurotrophic disturbance.

They also observed in two cases a questionable involvement of the colon in which the colon exhibited areas of increased rigidity between areas of sacculation.

This paper by Hale and Schatzki is of great importance because it calls attention to a hitherto unsuspected or not generally appreciated fact that the lesions of the gastrointestinal tract seen in patients suffering from scleroderma are not limited to the esophagus but are more widespread in the gastrointestinal tract, involving the small intestine and probably also the large bowel, and these gastrointestinal changes are of considerable clinical importance. The lesions of the esophagus simulate to some extent cardiospasm whereas those seen in the small intestine simulate ileus or deficiency disease and must be differentiated from them.



ALBERT FRANKLIN TYLER

1881—1944

ALBERT FRANKLIN TYLER, of Omaha, Nebraska, a member of the American Roentgen Ray Society since 1915, died on February 25, 1944, at the age of sixty-two. Truly a physician, memorialized by his own county society as having served his profession well indeed, Dr. Tyler died of coronary thrombosis. Several months earlier he had a series of coronary episodes, but he preferred to return to his work and take his chances rather than to have a possibly longer life of enforced inactivity. He died in Immanuel Hospital, where he was attending radiologist from 1915 on.

Dr. Tyler was born in Logan County, Illinois, on March 14, 1881. He was gradu-

ated from Nebraska Wesleyan University in 1904, and was later a trustee of this University, and from Creighton University Medical School in 1907. He was a member of the medical fraternity of Phi Rho Sigma, and of the honorary scholastic fraternity of Phi Kappa Phi. After serving an internship in the Omaha General Hospital, he took post-graduate studies at Johns Hopkins University Medical School and at the New York Post-Graduate Medical School.

He was clinical professor of radiology and physical therapy for many years at his Alma Mater. He was a founder of the American Congress of Physical Therapy; in 1932 he received its gold key award; in 1933 he was its president. He was a member of the American Radium Society and of the British Röntgen Society, a fellow of the American College of Physicians, and a past president of the Radiological Society of North America, in addition to the Omaha Douglas County Medical Society, the Nebraska State Medical Association, and the Nebraska State Radiological Society. He was a former associate editor of the *Nebraska State Medical Journal*, and editor and publisher of the *Journal of Radiology*, which later became the *Archives of Physical Therapy*. He contributed freely of his time, energy, and money to the History of Medicine in Nebraska, which he edited, and which was published in 1928.

Dr. Tyler is survived by his wife, who was Miss Charlotte Ellen Roe of Omaha, by a daughter, Mrs. Eugene Stromberg, of Ithaca, New York, and by a son, Edward Tyler, of Omaha.

JAMES F. KELLY

HENRY P. ENGELN

1870—1944

THE following Resolution of the Cleveland Radiological Society was ordered at the February, 1944 meeting:

WHEREAS: Death has removed from our Society, Mr. Henry P. Engeln, the following brief outline is submitted for incorporation in the records.

He was born in Paris, France, in 1870 and died at St. Luke's Hospital, Cleveland, Ohio on February 7, 1944. He came to the United States with his parents, when he was about fifteen years of age; they settled in Chicago, Illinois. There he grew to manhood and was in business with his brother, until he came to Cleveland, about the year 1900. Here he engaged in the manufacture of revolving plate static machines. This first brought him in touch with the x-ray field. He cooperated with Dr. George Iddings in establishing the first x-ray laboratory in Cleveland, which was located in the Caxton Building on Huron Road. He continued in this field the rest of his business life. He was associated with several leading x-ray organizations and for many years headed his own, the H. P. Engeln Co. He was always interested in everything pertaining to this branch of medicine. He regularly attended both national and international meetings; he was known to hundreds of radiologists and probably knew personally more pioneer radiologists than any other member. Even after retiring from active work a few years ago, he visited and enjoyed being with his old x-ray friends.

As a Society we wish to extend to his family our sympathy at his passing and assure them we hold fondly the memory of this friendly friend.

THEREFORE: Be it ordered that a copy of this Resolution be sent to the family, the AMERICAN JOURNAL OF ROENTGENOLOGY and *Radiology*.

SOCIETY PROCEEDINGS, CORRESPONDENCE AND NEWS ITEMS

Items for this section solicited promptly after the events to which they refer.

MEETINGS OF ROENTGEN SOCIETIES*

UNITED STATES OF AMERICA

AMERICAN ROENTGEN RAY SOCIETY
Secretary, Dr. H. Dabney Kerr, University Hospital, Iowa City, Iowa. Annual meeting: Joint Meeting of American Roentgen Ray Society and Radiological Society of North America, Palmer House, Chicago, Ill., Sept. 24–29, 1944.

AMERICAN COLLEGE OF RADIOLOGY
Secretary, Mac F. Cahal, 540 N. Michigan Ave., Chicago, Ill. Annual Meeting: Stevens Hotel, 10:00 A.M., June 14, 1944.

SECTION ON RADIOLOGY, AMERICAN MEDICAL ASSOCIATION
Secretary, Dr. J. T. Murphy, 421 Michigan St., Toledo, Ohio. Annual meeting: Chicago, Ill., June 12–16, 1944.

ARKANSAS RADIOLOGICAL SOCIETY
Secretary, Dr. J. S. Wilson, Mack Wilson Hospital, Monticello, Ark. Meets every three months and also at time and place of State Medical Association.

RADIOLOGICAL SOCIETY OF NORTH AMERICA
Secretary, Dr. D. S. Childs, 607 Medical Arts Bldg., Syracuse, N. Y. Annual meeting: Joint Meeting of American Roentgen Ray Society and Radiological Society of North America, Palmer House, Chicago, Ill., Sept. 24–29, 1944.

RADIOLOGICAL SECTION, BALTIMORE MEDICAL SOCIETY
Secretary, Dr. Walter L. Kilby, Baltimore. Meets third Tuesday each month, September to May.

SECTION ON RADIOLOGY, CALIFORNIA MEDICAL ASSOCIATION
Secretary, Dr. Earl R. Miller, University of California Hospital, San Francisco, Calif.

RADIOLOGICAL SECTION, CONNECTICUT MEDICAL SOCIETY
Secretary, Dr. Max Climan, 242 Trumbull St., Hartford, Conn. Meets bi-monthly on second Thursday, at place selected by Secretary. Annual meeting in May.

SECTION ON RADIOLOGY, ILLINOIS STATE MEDICAL SOCIETY
Secretary, Dr. H. W. Ackemann, 321 W. State St., Rockford, Ill.

RADIOLOGICAL SECTION, LOS ANGELES COUNTY MEDICAL ASSOCIATION
Secretary, Dr. Roy W. Johnson, 1407 S. Hope St., Los Angeles, Calif. Meets on second Wednesday of each month at the County Society Building.

RADIOLOGICAL SECTION, SOUTHERN MEDICAL ASSOCIATION
Secretary, Dr. Roy G. Giles, Temple, Texas.

BROOKLYN ROENTGEN RAY SOCIETY
Secretary, Dr. Leo Harrington, 880 Ocean Ave., Brooklyn, N.Y. Meets monthly on fourth Tuesday, October to April.

BUFFALO RADIOLOGICAL SOCIETY
Secretary, Dr. Joseph S. Gian-Francheschi, 610 Niagara St., Buffalo, N. Y. Meets second Monday of each month except during summer months.

CHICAGO ROENTGEN SOCIETY
Secretary, Dr. F. H. Squire, 1754 W. Congress St., Chicago 12, Ill. Meets second Thursday of each month October to April inclusive at the Palmer House.

CINCINNATI RADIOLOGICAL SOCIETY
Secretary, Dr. Samuel Brown, 707 Race St., Cincinnati, Ohio. Meets third Tuesday of each month, October to May, inclusive.

CLEVELAND RADIOLOGICAL SOCIETY
Secretary, Dr. D. D. Brannan, 11311 Shaker Blvd., Cleveland 4, Ohio. Meets at 6:30 P.M. at Allerton Hotel on fourth Monday each month, October to April, inclusive.

DALLAS-FORT WORTH ROENTGEN STUDY CLUB
Secretary, Dr. X. R. Hyce, Medical Arts Bldg., Fort Worth, Texas. Meetings held in Dallas on odd months and in Fort Worth on even months, on third Monday, at 7:30 P.M.

DENVER RADIOLOGICAL CLUB
Secretary, Dr. Edward J. Meister, 366 Metropolitan Bldg., Denver, Colo. Meets third Friday of each month at Denver Athletic Club.

DETROIT ROENTGEN RAY AND RADIUM SOCIETY
Secretary, Dr. E. R. Witwer, Harper Hospital. Meets monthly on first Thursday from October to May at Wayne County Medical Society Building.

FLORIDA RADIOLOGICAL SOCIETY
Acting Secretary, Dr. Walter A. Weed, 204 Exchange Bldg., Orlando, Fla. Meetings in May and November.

GEORGIA RADIOLOGICAL SOCIETY
Secretary, Dr. James J. Clark, 478 Peachtree St., Atlanta, Ga. Meets in November and at annual meeting of Medical Association of Georgia in the spring.

RADIOLOGICAL SOCIETY OF KANSAS CITY
Secretary, Dr. Arthur B. Smith, 800 Argyle Bldg., Kansas City, Mo. Meets third Thursday of each month at a place designated by the president.

ILLINOIS RADIOLOGICAL SOCIETY
Secretary, Dr. Wm. DeHollander, St. John's Hospital Springfield, Ill. Meets three times a year.

INDIANA ROENTGEN SOCIETY
Secretary, Dr. H. C. Ochsner, Methodist Hospital, Indianapolis. Meeting held the second Sunday in May annually.

KENTUCKY RADIOLOGICAL SOCIETY
Secretary, Dr. W. C. Martin, 321 W. Broadway, Louisville. Meets annually in Louisville on first Saturday in Apr.

LONG ISLAND RADIOLOGICAL SOCIETY
Secretary, Dr. Marcus Wiener, 1430-48th St., Brooklyn, N. Y. Meets Kings County Med. Soc. Bldg. monthly on fourth Thursday, October to May, 8:30 P.M.

LOUISIANA RADIOLOGICAL SOCIETY
Secretary, Dr. J. R. Anderson, 1130 Louisiana Ave., Shreveport. Meets annually during Louisiana State Medical Society Meeting.

MICHIGAN ASSOCIATION OF ROENTGENOLOGISTS
Secretary, Dr. E. M. Shebesta, 1429 David Whitney Bldg., Detroit. Three meetings a year, Fall, Winter, Spring.

MILWAUKEE ROENTGEN RAY SOCIETY
Secretary, Dr. C. A. H. Fortier, 231 W. Wisconsin Ave., Milwaukee, Wis. Meets monthly on second Monday at University Club.

MINNESOTA RADIOLOGICAL SOCIETY
Secretary, Dr. Annette T. Stenstrom, 1218 Medical Arts Bldg., Minneapolis, Minn. One meeting a year at time of Minnesota State Medical Association.

NEBRASKA RADIOLOGICAL SOCIETY
Secretary, Dr. D. A. Dowell, Medical Arts Bldg. Omaha, Nebr. Meets third Wednesday of each month, at 6 P.M. at either Omaha or Lincoln.

NEW ENGLAND ROENTGEN RAY SOCIETY
Secretary, Dr. Hugh F. Hare, Lahey Clinic, Boston, Mass. Meets monthly on third Friday, Boston Medical Library.

RADIOLOGICAL SOCIETY OF NEW JERSEY
Secretary, Dr. H. J. Perlberg, 921 Bergen Ave., Jersey City. Meets annually at time and place of State Medical Society. Mid-year meetings at place chosen by president.

NEW YORK ROENTGEN SOCIETY
Secretary, Dr. Ramsay Spillman, 115 East 61st St., New York City. Meets monthly on third Monday, New York Academy of Medicine, at 8:00 P.M.

NORTH CAROLINA ROENTGEN RAY SOCIETY
Secretary, Dr. Major Fleming, Rocky Mount, N. C. Annual meeting at time and place of State Medical Society. Mid-year scientific meeting at place designated.

* Secretaries of Societies not here listed are requested to send the necessary information to the Editor.

NORTH DAKOTA RADIOLOGICAL SOCIETY
Secretary, Dr. L. A. Nash, St. John's Hospital, Fargo. Meetings held by announcement.

CENTRAL NEW YORK ROENTGEN RAY SOCIETY
Secretary, Dr. C. F. Potter, 820 S. Crouse Ave., Syracuse. Three meetings a year. January, May, November.

OHIO RADIOLOGICAL SOCIETY
Secretary, Dr. J. E. McCarthy, 707 Race St., Cincinnati. Meets at time and place of annual meeting of Ohio State Medical Association.

PACIFIC ROENTGEN SOCIETY
Secretary, Dr. L. H. Garland, 450 Sutter St., San Francisco, Calif. Meets annually, during meeting of California Medical Association.

PENNSYLVANIA RADIOLOGICAL SOCIETY
Secretary, Dr. L. E. Wurster, 416 Pine St., Williamsport. Annual meeting at Penn Alto Hotel, Altoona, Pa., May 13–14, 1944.

PHILADELPHIA ROENTGEN RAY SOCIETY
Secretary, Dr. R. P. Barden, University Hospital, Meetings first Thursday of each month from October to May inclusive at 8:15 P.M., in Thompson Hall, College of Physicians, 19 S. 22d St.

PITTSBURGH ROENTGEN SOCIETY
Secretary, Dr. R. G. Alley, 4800 Friendship Ave. Meets second Wednesday each month, 4:30 P.M., October to June, Pittsburgh Academy of Medicine.

ROCHESTER ROENTGEN RAY SOCIETY, ROCHESTER, N. Y.
Secretary, Dr. Sidney Larson, Strong Memorial Hospital. Meets monthly on third Monday from October to May, inclusive, 8 P.M. at Strong Memorial Hospital.

ROCKY MOUNTAIN RADIOLOGICAL SOCIETY
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Secretary, Dr. Henry L. Jaffe, Naval Hospital, Balboa Park, San Diego, Calif. Meets monthly on first Wednesday at dinner.

SAN FRANCISCO RADIOLOGICAL SOCIETY
Secretary, Dr. Martha Mottram, 450 Sutter St., San Francisco. Meets monthly on third Thursday at 7:45 P.M., first six months of year at Toland Hall, University of California Hospital, second six months at Lane Hall, Stanford University Hospital.

SHREVEPORT RADIOLOGICAL CLUB
Secretary, Dr. R. W. Cooper, Charity Hospital, Shreveport, La. Meets monthly on third Wednesday, at 7:30 P.M., September to May inclusive.

SOUTH CAROLINA X-RAY SOCIETY
Secretary, Dr. T. A. Pitts, Baptist Hospital, Columbia, S. C. Meets in Charleston on first Thursday in November, also at the time and place of South Carolina State Medical Association.

TENNESSEE RADIOLOGICAL SOCIETY
Secretary, Dr. J. M. Frère, 707 Walnut St., Chattanooga, Tenn. Meets annually at the time and place of the Tennessee State Medical Association.

TEXAS RADIOLOGICAL SOCIETY
Secretary, Dr. Herman Klapproth, Sherman, Texas.

UNIVERSITY OF MICHIGAN DEPARTMENT OF ROENTGENOLOGY STAFF MEETING
Meets each Monday evening from September to June, at 7 P.M. at University Hospital.

UNIVERSITY OF WISCONSIN RADIOLOGICAL CONFERENCE
Secretary, Dr. E. A. Pohle, 1300 University Ave., Madison, Wis. Meets every Thursday from 4:00–5:00 P.M. Room 301, Service Memorial Institute.

VIRGINIA RADIOLOGICAL SOCIETY
Secretary, Dr. E. L. Flanagan, 116 E. Franklin St., Richmond, Va. Meets annually in October.

WASHINGTON STATE RADIOLOGICAL SOCIETY
Secretary, Dr. Thomas Carlile, 1115 Terry St., Seattle. Meets fourth Monday each month, October through May, College Club, Seattle.

X-RAY STUDY CLUB OF SAN FRANCISCO
Secretary, Dr. J. M. Robinson, University of California Hospital. Meets monthly in evening on third Thursday.

CUBA

SOCIEDAD DE RADIOLOGÍA Y FISIOTERAPIA DE CUBA
President, Dr. J. Manuel Viamonte, Hospital Mercedes, Habana, Cuba. Meets monthly in Habana.

BRITISH EMPIRE

BRITISH INSTITUTE OF RADIOLOGY INCORPORATED WITH THE RÖNTGEN SOCIETY
Medical Members' meeting held monthly on third Friday at 5:30 P.M. and Ordinary Meeting at same time on following Saturday, October to May, 32 Welbeck St., London, W.1.

SECTION OF RADIOLOGY OF THE ROYAL SOCIETY OF MEDICINE (CONFINED TO MEDICAL MEMBERS)
Meets on the third Friday of each month at 4:45 P.M. at the Royal Society of Medicine 1, Wimpole St., London, W. 1.

FACULTY OF RADIOLOGISTS
Secretary, Dr. M. H. Jupe, 32 Welbeck St., London, W. 1 England.

SECTION OF RADIOLOGY AND MEDICAL ELECTRICITY, AUSTRALASIAN MEDICAL CONGRESS
Secretary, Dr. H. M. Cutler, 139 Macquarie St., Sydney, New South Wales.

RADIOLOGICAL SECTION OF THE VICTORIAN BRANCH OF THE BRITISH MEDICAL ASSOCIATION
Secretary, Dr. Keith Hallam, St. George's Hospital, E.E.W., Melbourne, E. 4, Victoria, Australia. Meets monthly from March to November inclusive.

CANADIAN ASSOCIATION OF RADIOLOGISTS
Secretary, Dr. A. D. Irvine, 540 Tegler Bldg., Edmonton, Alberta.

SECTION OF RADIOLOGY, CANADIAN MEDICAL ASSOCIATION
Secretary, Dr. C. M. Jones, Inglis St., Ext. Halifax, N. S.

RADIOLOGICAL SECTION, NEW ZEALAND BRITISH MEDICAL ASSOCIATION
Secretary, Dr. Colin Anderson, Invercargill, New Zealand. Meets annually.

SOUTH AMERICA

SOCIEDAD ARGENTINA DE RADIOLOGIA
Secretary, Dr. Guido Gotta, Buenos Aires, Argentina. Meetings are held monthly.

CONTINENTAL EUROPE

SOCIEDAD ESPANOLA DE RADIOLOGIA Y ELECTROLOGIA
Secretary, Dr. J. Martin-Crespo, Fuencarral, 7. Madrid, Spain. Meets monthly in Madrid.

SOCIÉTÉ SUISSE DE RADIOLOGIE (SCHWEIZERISCHE RÖNTGEN-GESELLSCHAFT)
Secretary for French language, Dr. A. Grosjean La Chaux de Fonds.
Secretary for German language, Dr. Scheurer, Molzgasse Bern. Meets annually in different cities.

SOCIETATEA ROMANA DE RADIOLOGIE SI ELECTROLOGIE
Secretary, Dr. Oscar Meller, Str. Banul Mărăcine, 30, S. I., Bucuresti, Roumania. Meets second Monday in every month with the exception of July and August.

ALL-RUSSIAN ROENTGEN RAY ASSOCIATION, LENINGRAD: USSR in the State Institute of Roentgenology and Radiology, 6 Roentgen St.
Secretaries, Drs. S. A. Reinberg and S. G. Simonson. Meets annually.

LENINGRAD ROENTGEN RAY SOCIETY
Secretaries, Drs. S. G. Simonson and G. A. Gusterin. Meets monthly, first Monday at 8 o'clock, State Institute of Roentgenology and Radiology, Leningrad.

MOSCOW ROENTGEN RAY SOCIETY
Secretaries, Drs. L. L. Holst, A. W. Ssamygin and S. T. Konobejevsky. Meets monthly, first Monday, 8 P.M.

SCANDINAVIAN ROENTGEN SOCIETIES
The Scandinavian roentgen societies have formed a joint association called the Northern Association for Medical Radiology, meeting every second year in the different countries belonging to the Association.

AMERICAN COLLEGE OF RADIOLOGY

The Annual Meeting of the American College of Radiology will be held at the Stevens Hotel at 10:00 A.M. on Wednesday, June 14, 1944, during the week of the meeting of the American Medical Association. Fellows and members who are planning to attend should make hotel reservations immediately through the Housing Committee of the American Medical Association.

JOURNAL OF NEUROSURGERY

The new *Journal of Neurosurgery* which is to be published bimonthly for the Harvey Cushing Society, has, with the first issue, established itself proudly among the current medical journals. The hitherto hybrid neurosurgeon now has a journal of his own in which to publish his own peculiar triumphs, trials and theories. The well balanced selection of papers for the first issue is a tribute to the editorial management of Louise Eisenhardt. Dr. Cushing would indeed have been pleased with the varied contributions of his friends and pupils in the new journal and no doubt would even have smiled tolerantly at the name—*Journal of Neurosurgery*—his own preference being the term neurological surgery.

CHICAGO ROENTGEN SOCIETY

At the annual meeting of the Chicago Roentgen Society held on Thursday, April 13, 1944, the following officers were elected for the coming year: *President*, Dr. Warren W. Furey; *Vice-President*, Dr. T. J. Wachowski; *Secretary-Treasurer*, Dr. Fay H. Squire; *Trustees*, Dr. Earl E. Barth, Dr. Frank L. Hussey and Dr. Benjamin D. Braun.

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ABSTRACTS OF ROENTGEN AND RADIUM LITERATURE

ROENTGEN DIAGNOSIS

HEAD

PUDENZ, ROBERT H. Repair of cranial defects with tantalum; experimental study. *J. Am. M. Ass.*, Feb. 13, 1943, *121*, 478–481.

Enthusiasm for the employment of the alloplastic materials has recently been revived by the encouraging reports of Geib, Beck and Peyton and Hall on the repair of cranial defects with vitallium. In this paper certain observations on the experimental and clinical use of tantalum for this purpose are reported.

Tantalum plates were implanted in the cranial defects of 11 cats. The operative procedures were carried out under sterile conditions using intraperitoneal soluble pentobarbital anesthesia. The animals were killed after periods of survival varying from twenty-seven to 318 days. There was no evidence of reaction to the metal during the observation period in any of the animals.

Results. In all the experiments the tantalum plates were enveloped in delicate translucent layers of connective tissue which were continuous with the pericranium. The capsule was completely formed at the end of twenty-seven days and showed no tendency to progressive thickening. Even at the end of 318 days, the bluish white luster of the metal was easily perceptible through the delicate layers of the capsule. These layers were tightly adherent to the tantalum plates and intracapsular fluid formation was absent. The inner layer was adherent to the dura but easily separable from it. When the capsules were opened the lining was invariably found to be smooth and glistening. Osteoblastic activity was not hindered, and even at the end of twenty-seven days the proliferating bone at the edge of the defect had closely approximated the edge of the metal. In the animals surviving 317 and 318 days there was a complete closure of the cranial defect by the new bone formation. Without exception, the tantalum plates retained their original luster and showed no signs of corrosion. An inflammatory reaction leading to extrusion or necessitating removal of the plates did not occur.—*S. G. Henderson.*

KAHN, EDGAR A. Contrast media in cysts and abscesses of the cerebral hemisphere. *Surg., Gynec. & Obst.*, May, 1942, *74*, 785–790.

It has been the experience of all neurosurgeons in attempting ventricular air injection or biopsy of a cerebral tumor to enter unexpectedly a cystic cavity with the exploring needle, xanthochromic fluid or pus being obtained. To approach such a lesion in the most direct manner and thus simplify operative procedure, the use of a contrast medium is desirable. Air and iodized poppy seed oil have been used for this purpose but they do not mix with fluid contents. Sodium iodide is miscible but colloidal thorium dioxide (thorotrast) is less irritating, mixes readily and has the added advantage of being phagocytosed by the cellular elements of an abscess wall, making the capsule radiopaque.

Histories of cases are presented in an effort to show the value of colloidal thorium dioxide cystography. In an astrocytoma in a fourteen year old girl, the cyst was outlined showing the mural nodule attached to the anterior wall. Cushing long ago showed that if the mural nodule of an astrocytoma were removed the cyst wall need not be dealt with. Thus, in this case an effective operation was carried out by means of a comparatively simple procedure.

The author suggests that there might be wider application in the field of general surgery as in visualization of an empyema cavity by means of the thorotrast. In the drainage of a thoracic empyema, the difficulty increases as the cavity becomes smaller. It is at this later stage that the colloidal thorium dioxide which has been phagocytosed by the empyema wall might show when obliteration is complete.—*Mary Frances Vastine.*

NECK AND CHEST

DOCKERTY, MALCOLM B., and MAYO, CHARLES W. Primary tumors of the submaxillary gland with special reference to mixed tumors. *Surg., Gynec. & Obst.*, June, 1942, *74*, 1035–1041.

Eighty-one primary submaxillary malignant tumors have been reviewed from the clinical

and pathological aspects. The results of the study are as follows:

1. True mixed tumors constituted 63 per cent of the series. They represented low grade, slowly growing adenocarcinomas usually not associated clinically with pain or local fixation. The tendency toward recurrence was not pronounced except when inadequate "tissue sacrifice" had been carried out.

2. Adenocarcinomas of the cylindroma type accounted for 18.5 per cent of the tumors. These cylindromas, as a rule, were associated with a short clinical history in which pain and local fixation were prominent features. Pathologically the lesions, although they were of a moderate degree of malignancy, presented pronounced infiltrative tendencies with selective invasion of nerves. The rate of recurrence was extremely high and the outlook generally unfavorable.

3. An "intermediate" group of cases was found in which the tumors pathologically shared the features of the mixed tumors and the cylindromas. Clinically, also, the histories of the patients justified separation of the tumors into a special category.

4. Eight patients, 9.5 per cent, had atypical tumors described as "miscellaneous." Prognosis depended partly on the type and grade of the lesion and partly on the extent of lymph node involvement.

5. Clinical follow-up studies have demonstrated the usefulness of the microscopic classification presented by the authors in relation to prognosis.

6. Throughout the study, the authors were impressed by the disappointing results which followed conservative operation on malignant tumors of the submaxillary gland.—*Mary Frances Vastine.*

WARREN, GEORGE A. Amyloid goiter. *Surg., Gynec. & Obst.*, Sept., 1942, *75*, 374–378.

There have been reports of 56 cases of amyloid goiter in the literature. The author reports 2 additional cases bringing the total to 58. The following conclusions have been made:

1. The commonest primary disease associated with amyloid infiltration of the thyroid is pulmonary tuberculosis. Purulent bronchitis with bronchiectasis or neoplastic processes are less commonly the precursors.

2. Amyloid infiltration of the thyroid may occur as a part of a primary amyloidosis.

3. Amyloid infiltration of the thyroid without amyloid in any other organ is a very rare phenomenon, only 2 such cases being found in the literature.

4. Amyloid infiltration of the thyroid gland causes enlargement and an increase in consistency in a majority of the cases.

5. Amyloid infiltration of the thyroid is sometimes associated with fat tissue infiltration as well. There is the possibility that this is more than a casual relationship.

6. In no case of thyroid amyloidosis has there been any evidence of thyroid insufficiency.

7. The most common error in diagnosis is to conclude that a malignancy is present when actually amyloid is responsible for the enlargement, increase in consistency and nodularity of the thyroid.—*Mary Frances Vastine.*

KING, WILLIAM L. M., and PEMBERTON, J. DEJ. So called lateral aberrant thyroid tumors. *Surg., Gynec. & Obst.*, May, 1942, *74*, 991–1001.

Fifty-four cases in which a benign or a malignant tumor of thyroid tissue was discovered in the lateral region of the neck without any gross connection with the thyroid gland itself are analyzed. The following conclusions are reached:

1. So-called lateral aberrant thyroid tumors are nearly always metastatic extensions to the deep cervical lymph nodes from primary carcinoma in the homolateral lobe of the thyroid gland.

2. Seventy-four per cent of these tumors are papillary adenocarcinomas and spread by lymphatic extension.

3. Sixty per cent of "lateral aberrant thyroid tumors" are associated with thyroid tumors of identical structure which are always situated on the same side.

4. The "aberrant" tumors should be treated by radical block dissection of the neck and removal of the corresponding thyroid lobe whether or not a tumor can be felt or seen in that lobe at the operating table.

5. Complete removal of all nodes and thyroid tissue known to be involved is essential if recurrence is to be prevented. Treatment by radiation is of questionable benefit unless surgical removal of the diseased tissue has been complete.

6. Following complete surgical removal the prognosis is good in cases of papillary and low grade non-papillary carcinomas because the rate of growth of these lesions is very slow and these tumors do not commonly invade sur-

rounding structures; in the inoperable or neglected cases, they may have little effect on the patient's general health for years but they may eventually cause death.—*Mary Frances Vastine.*

ELKELES, A. A case of the Plummer-Vinson syndrome with radiologically demonstrable peptic ulcer of the oesophagus. *Brit. J. Radiol.*, April, 1942, *15*, 122–123.

Dysphagia associated with hypochromic microcytic anemia constitutes the so-called Plummer-Vinson syndrome. Roentgen examinations have shown that the dysphagia is not functional, as was at first believed, but that there are organic changes consisting of atrophic changes in the mouth, glossitis and a post-cricoid web.

A case is described in a woman of fifty-two and illustrated with a roentgenogram. In addition to the above-mentioned changes, there were spasm and niche formation at the cardiac end of the esophagus, indicating peptic ulcer. The blood count showed pernicious anemia and stomach examination showed achlorhydria. She improved on liver injections and iron.

It is probable that, in addition to the ordinary type of pernicious anemia, there is a type that develops from hypochromic microcytic anemia and the dysphagia in these cases may be due, partly at least, to a deficiency of iron. No case of peptic ulcer at the cardiac end of the esophagus has been reported previously in the Plummer-Vinson syndrome. This case shows the advisability of making a careful examination of the whole esophagus.

It is possible that the organic changes in this syndrome may be precancerous, in analogy with the findings in chronic gastritis with achlorhydria.—*Audrey G. Morgan.*

SPARKS, J. V., and SCRIMGEOUR, D. MUIR. Transposition of the aortic arch; report of three cases. *Brit. J. Radiol.*, May, 1942, *15*, 150–153.

Three cases of transposition of the aortic arch are described and illustrated with roentgenograms. This abnormality is very rare. In the first 2 cases the aortic arch developed from the fourth right arterial arch and an aortic diverticulum formed at the termination of the arch on the left. In the third case the aortic knuckle shadow was prominent to the right of the sternum.

This is the easiest of all the congenital anomalies of the circulatory system to diagnose and the importance of recognizing it is illustrated by a case described by Dr. Hope Gosse in which he was able to prevent a patient from being treated by irradiation for a mediastinal tumor and by Case III of this group in which an exploratory thoracotomy had been carried out before the patient was examined by the authors.—*Audrey G. Morgan.*

BARCLAY, A. E., FRANKLIN, K. J., and PRICHARD, M. M. L. The mechanism of closure of the ductus venosus. *Brit. J. Radiol.*, March, 1942, *15*, 66–71.

The authors studied the closure of the ductus venosus on mature lambs obtained by cesarean section. Diagrammatic sketches and roentgenograms of the region are given illustrating their findings. They used direct and indirect roentgen cinematography combined with intravascular injections of radiopaque medium made into one of the veins of the umbilical cord. They found roentgen evidence of a sphincter or similar structure at the beginning of the ductus venosus and concluded that this structure must bring about the functional closure of the ductus. They then made dissections and found anatomical evidence also of the existence of such a structure. The question is now being studied by two histologists, one in England and one in the United States, and so far their results are the same and justify the authors' conclusion that the smooth muscle at the beginning of the ductus venosus is concerned with the functional closure of the channel. Their report will be published later.—*Audrey G. Morgan.*

TUCKER, WILLIAM B., and BRYANT, J. E. The problem of unsuspected tuberculosis during pregnancy in the Negro; incidence by roentgenologic techniques in 1,000 consecutive cases. *Am. J. Obst. & Gynec.*, April, 1941, *45*, 678–685.

One thousand consecutive patients attending the Prenatal Clinic of the Provident Hospital and Training School in Chicago were examined for pulmonary tuberculosis by roentgenoscopy and roentgenography. (1) Eighteen cases (1.8 per cent) of unsuspected clinically important tuberculosis and 11 additional cases (1.1 per cent) of clinically unimportant reinfection tuberculosis, a total of 29 cases (2.9 per cent), were found. This incidence was found to be approximately the same as for other non-pregnant women of the same age groups examined at the Provident Hospital. (2) The incidence of un-

suspected clinically important tuberculosis at the Chicago Lying-in Hospital was found to be 1.0 per cent by Eisele *et al.* The difference in the figures between the two hospitals is probably due to the different economic and social status of the two groups rather than to race or a slight difference in the age distribution between the two groups. (3) The findings serve to confirm the contention that routine chest roentgenologic examinations should be an indispensable part of the prenatal care of pregnant women.—*Mary Frances Vastine.*

HORSLEY, J. SHELTON. Pulsating tumors of the anterior mediastinum. *Surg., Gynec. & Obst.*, July, 1942, *75*, 49–53.

A case is reported in which a tumor was removed from the anterior mediastinum behind the upper manubrium. Pulsation evidently transmitted from the aorta through the tumor had eroded the upper sternum. The defect was smooth in outline. The tumor was thought to be malignant and probably a metastatic hypernephroma. However, a primary lesion could not be found and the patient was found to be perfectly well at the last follow-up two years after the removal of the mass.

A neoplasm arising in the anterior mediastinum and eroding through the sternum by transmitted pulsation from the aorta is extremely rare. Crile has reviewed 13 cases of pulsating neoplasms of the sternum and has added 5 from the records of the Cleveland Clinic. Of this group, 9 were probably cases of metastatic hypernephroma and 9 were probably metastases from malignant adenoma of the thyroid.

The possibility of this lesion being a Pancoast tumor is discussed. These tumors are located in the posterior thoracic inlet adjacent to the pulmonary apex (the superior pulmonary sulcus). The site of origin in independent of lung, pleura, ribs, vertebrae, mediastinum or metastatic foci. Pancoast believed that they arose from a persistent rest from the ectodermal portion of the branchial structures. Horner's syndrome is produced. (This should really be called Hare's syndrome since Hare described this symptom complex thirty-one years before Horner.) There is pain which is referred to the shoulder and arm associated with muscle atrophy. The roentgenogram shows a small homogeneous apical shadow and is suggestive of destructive infiltration of contiguous ribs and vertebrae.—*Mary Frances Vastine.*

HEAD, JEROME, AND HUDSON, THEODORE R. Subphrenic abscess with bronchial fistula. *Surg., Gynec. & Obst.*, July, 1942, *75*, 54–60.

It is observed that hepatic, subdiaphragmatic and perinephritic abscesses frequently perforate the diaphragm and are evacuated by expectoration. Collections of pus in the upper abdomen frequently burrow upward through the diaphragm whereas similar collections in the thorax rarely, if ever, extend downward into the abdomen. The incidence of perforation of the diaphragm is so high in subdiaphragmatic abscess that this complication is to be expected when operation is too long delayed.

Next to the history, the roentgenogram gives the most important diagnostic information in this condition. The most characteristic finding is a greater or lesser elevation of the diaphragm with air and a fluid level beneath it. Neither the elevation nor the air and the fluid level are always present. There may be no more than evidences of old pleurisy at the base. The findings in the lungs and pleura above the diaphragm vary greatly. The perforation may have produced an empyema as well as a bronchial fistula. In acute cases there may be extensive bronchopneumonia in the lower lobe on the affected side. In more chronic cases, areas of chronic pneumonitis may be present in one or both lungs. Not infrequently pulmonary and pleural findings are a most entirely absent, consisting merely of increased peribronchial markings extending into one lower lobe. The meagerness of thoracic findings in the presence of fever and copious expectoration is one of the important diagnostic points. In such cases it is necessary to employ bronchography to rule out bronchiectasis. However, when this has been done and one is still unable to explain the abundant expectoration on the basis of revealed pulmonary or pleural pathema, it is well to consider the possibility that the pus is coming from below the diaphragm.

Roentgenograms taken after the injection of 300 to 400 cc. of air into the peritoneal cavity are of great value. Under normal conditions with the patient in the upright position, these will reveal air beneath both halves of the diaphragm. In the presence of subdiaphragmatic abscess, no air can penetrate into this space on the affected side. If the under surfaces of the diaphragm are free, pus in this region can be ruled out.—*Mary Frances Vastine.*

Gosse, A. Hope. A case of hydatid disease of the lungs. *Brit. J. Radiol.*, March, 1942, *15*, 92–93.

A case of roentgen and laboratory diagnosis of hydatid cyst of both lungs is reported which is considered interesting because of the rarity of the disease in England. The patient was a laborer, aged thirty-two, who came for examination on account of a history of hemoptysis. In March, 1939, he was treated for bronchopneumonia and from the roentgenogram made at that time a diagnosis of abscesses of the lungs was made. Apparently at that time he had a cyst at the base of each lung. When he came to the hospital in 1941 because of hemoptysis, the roentgenogram showed a circular shadow at the left base of homogeneous density surrounded by normal lung tissue. At the right base there was infiltration, some fibrosis and a small area suggestive of loss of lung tissue. This roentgenogram is reproduced. Examination of the sputum showed a fragment of white membrane about the size of a postage stamp which evidently came from the lining of a cyst. The patient said he had coughed up bits like that for a week but never before that.

The diagnosis of hydatid cyst of the left lung and cyst of the right lung ruptured into a bronchus was based on the two roentgenograms, the presence and appearance of the expectorated membrane, an eosinophilia of 8 per cent, the positive intradermal Casoni reaction and ten years' residence in India.—*Audrey G. Morgan.*

Winn, William A. Pulmonary cavitation associated with coccidioidal infection. *Arch. Int. Med.*, Dec., 1941, *68*, 1179–1214.

Cavitation heightens the close similarity between the roentgen appearances of coccidioidal and tuberculous pulmonary infections; without identification of the causative organism diagnostic confusion may occur. Coccidioidal cavitation usually develops during, or shortly after, the primary stage of pulmonary infection; it is far more benign than tuberculous cavitation as demonstrated in the 13 cases reported in this paper. In cases of primary coccidioidal cavitation the prognosis is entirely favorable; prolonged institutional care is unnecessary; early return to usual occupation despite persistence of a pathologically latent cavity is permitted; definitive collapse procedures to close such cavities are avoided except for possible performance of pneumothorax if hemoptysis is frequent or massive or if the cavitation exhibits a more active phase than the thin-walled cavities described here.

The acute infection is *not* transmitted from person to person via sputum containing the endospores; hence, patients with coccidioidal infection are not considered a menace to society. The disease is endemic in the San Joaquin Valley and near-by valleys in central California, parts of Arizona and Texas and the Chaco regions of Argentina. In such areas the infection is widespread, as indicated by positive skin reactions to coccidioidin by a large proportion of San Joaquin Valley residents. Evidence of such widespread infection is difficult to obtain by an ordinary roentgen examination of the chest, with probable exception of calcification.

Residual pulmonary cavitation is regarded as only an infrequent manifestation of the primary coccidioidal infection, although in an unknown number of cases these cavities close spontaneously. Coccidioidal granuloma remains a relatively uncommon disease; pulmonary cavitation associated with it is unusual and should be easily distinguishable from the cavitation in primary coccidioidal infection. In the latter, severe constitutional symptoms are strikingly absent despite the existence of pulmonary cavitation, hemoptysis or spherule-laden sputum.

The primary infection by *Coccidioides immitis* is commonly accompanied by symptoms of bronchitis with a slightly productive cough, malaise, anorexia, chills, fever, headache, backache, night sweats and pleurisy (organisms may be recovered from the occasional effusion as well as from the sputum). Roentgen studies of the chest "may reveal only changes in the area of the lung root, such as peribronchial haziness, or there may be definite patches of pneumonitis, which often assumes a nodular form. Occasionally, exudative lesions involving lobules, or even entire lobes of the lung, are present and may be associated with early cavitation. Recovery is followed by a walling off or clearing of the primary focus without complication. Evidence of the first infection may remain and be revealed in the roentgenogram in the form of localized caseous or calcified foci or cavitation." Cutaneous sensitivity to coccidioidin develops seven to fourteen days after such infection; erythema nodosum or multiforme occurs in 2 to 5 per cent of cases. Primary infection with few or no symptoms also occurs.

Residual coccidioidal pulmonary cavitation may be asymptomatic after the acute primary

infection has subsided. Cavitation in conjunction with the acute primary infection alters the above clinical picture very little; most of such patients do give a history of hemoptysis, however. The term "latent" has been applied to residual cavities to indicate that the process is no longer progressive. "Healed" would be a misnomer since these cavities harbor viable organisms of *C. immitis*. Criteria for the diagnosis of latent cavitation were based on roentgenographic appearance of a thin-walled cavity, which appeared "punched out" or "dead," with little or no surrounding collateral reaction. A history of hemoptysis, lower respiratory tract infection and slightly productive cough was usually obtained. Intracutaneous coccidioidin skin tests yielded positive results; sputum or gastric content contained *C. immitis*. In no case did follow-up (few months to several years) studies reveal dissemination or progression of the disease.

The latent coccidioidal cavities are "considered as more or less benign, stabilized structures which are the end-results of a small focus of destruction produced by the primary coccidioidal infection." Bronchial communications may be large or small. Detailed case reports with associated roentgenographic studies of 12 cases of latent cavitation and 1 case of bronchiectasis due to coccidioidal infection are included.—*Henry G. Moehring.*

KERLEY, PETER. Wounds of the diaphragm. *Brit. J. Radiol.*, April, 1942, *15*, 97–99.

Diaphragmatic hernia is a frequent sequela to gunshot wounds of the thorax and abdomen. Many of these cases were overlooked in the last war. A case is described which suggests that the diagnosis can be made by careful roentgen examination.

The patient was a man, aged thirty-five, admitted to hospital fourteen hours after being injured by a bomb. There was a wound of the head and one of the chest with fracture of the sixth left rib posteriorly and a large bomb fragment in the lower lobe of the partially collapsed left lung. The whole of the lung was relatively opaque, indicating hemorrhagic atelectasis. Suspicion of an injury of the diaphragm was raised by the absence of a fluid level and of a clear diaphragmatic shadow. Operation showed that the diaphragm had been torn. The missile was extracted and the lung and diaphragm sutured. After a period of bronchopneumonia the patient recovered.

The roentgen findings which indicate injury of the peritoneum are: invisibility of the injured dome; intermittent twitching movements visible on the screen; the presence of a pneumoperitoneum if the wound is thoracic; an extensive wound of the lung with hemorrhagic atelectasis and little blood in the pleural cavity.

As these patients are usually most comfortable in the semi-recumbent position much of the blood drains into the abdominal cavity, which accounts for the absence of a fluid level. —*Audrey G. Morgan.*

RIVES, JAMES D. Anatomy and surgery of diaphragmatic hernia. Editorial. *Surg., Gynec. & Obst.*, May, 1942, *74*, 1026–1028.

The diaphragm is something of an afterthought in the evolutionary process, being present as a complete septum only in mammals. The major part of it is assembled from three ill-assorted structures—the septum transversum, the pleuroperitoneal membranes, and the dorsal mesogastrium. These structures descend from the cephalic portion of the celomic cavity on different time schedules and finally unite to form a single complete septum between the abdominal and thoracic cavities at about the seventh or eighth week of fetal life. Obviously, any disturbance of the orderly progress of this intricate process may result in the formation of defects in the diaphragm.

The phrenic nerve develops and descends with the segment formed from the septum transversum and invades the remainder of the diaphragm after fusion of the three segments. If this invasion is arrested prematurely, the musculature of that part of the diaphragm that has not received a nerve supply probably undergoes atrophy, leaving only the peritoneum and the pleura to separate the two great body cavities in that particular location.

Soon after the diaphragm becomes a complete septum, the mid-portion of the celomic cavity is rapidly expanded by growth of the liver, and the resulting tension of the diaphragm causes it to pick up from the body wall a supplementary rim of tissue. One would, therefore, expect the diaphragm to be continuous with the inner layer of the abdominal cavity. Recent anatomical studies by Baker have shown that this is actually the case. The anterior and lateral portions of the diaphragm are continuous with the transversus abdominis muscles and the posterior sheath of the rectus muscles. The attachment of these structures to the costal

arch is by means of what is, in effect, a common aponeurosis. Likewise the posterior or lumbar segments of the diaphragm are continuous with the anterior layer of the lumbodorsal fascia, except for the central portion, which is attached to the bodies of the lumbar vertebrae.—*Mary Frances Vastine.*

ABDOMEN

CORDINER, G. R. M. Duodenitis. *Brit. J. Radiol.*, Feb., 1942, *15*, 33–38.

Until recently chronic duodenitis has not been recognized as a clinical entity. Kirklin was the first to describe the roentgen appearance of this disease in a series of 32 cases which were proved histologically. His signs, however, may be absent in frank cases of duodenitis and the direct roentgen signs based on examination of the mucosal relief are of prime importance in the diagnosis of the disease.

One of the most important signs is broadening and thickening of the mucosal folds with a corresponding broadening of the hollows between them. To justify a diagnosis of duodenitis the folds must also show changes in their consistency. They show loss of flexibility, decreased movement on palpation and are less easily smoothed out by pressure and stretching. Sometimes the folds are so stiff that they appear to be rigid anatomical structures. The stiffening resembles that which takes place in a starched garment. The caliber of the swollen and stiffened folds is not uniform. But in some cases the relief pattern consists of fine, irregular elevations and depressions which are easily smoothed out by compression. The changes, which are generally localized in the cap, may extend into the descending portion of the duodenum. As a rule, erosions cannot be detected on the roentgenogram.

Roentgenograms are given showing the normal appearance of the duodenal mucosa and the various pictures seen in duodenitis.—*Audrey G. Morgan.*

WOLFER, JOHN A., BEATON, LINDSAY E., and ANSON, BARRY J. Volvulus of the cecum. *Surg., Gynec. & Obst.*, April, 1942, *74*, 882–894.

Over 300 cases of volvulus of the cecum have been reported in the international literature. This is a condition, therefore, which should be kept in mind in the differential diagnosis of disease presenting in the right lower quadrant of the abdomen and in the differential diagnosis of intestinal obstruction.

Volvulus of the cecum is defined as torsion limited to the cecum and adjacent terminal ileum and ascending colon.

The authors studied 125 normal adult cadavers consecutively and made the following observations: (1) In 11.2 per cent the cecum is mobile enough to allow the development of volvulus. (2) The anatomical arrangement which best predisposes to cecal volvulus is believed to be one in which the cecum is free and the ascending colon is fixed somewhere along its vertical course.

Incidence of Volvulus. Volvulus is most prone to occur in males in the twenty to forty year age group.

Etiology of Volvulus. The inciting agent of this disease may be purely mechanical or may be due to as yet incompletely understood physiological faults in the complex functioning of the right colon.

A case of cecal volulus is reported. A study of this case serves to emphasize the necessity for prompt surgical care and the importance of early examination by roentgenographic means.—*Mary Frances Vastine.*

BERNE, CLARENCE J. Diagnosis and treatment of amebic liver abscess. *Surg., Gynec. & Obst.*, Aug., 1942, *75*, 235–238.

In 1931 it was found that the mortality in amebic liver abscess at the Los Angeles County Hospital was 85 per cent. Since 1931, 74 cases of amebic abscess have been recognized and these cases furnish the basis for this report.

Diagnosis. (1) Amebic infection of the colon always antedates the liver lesion but many cases of amebic colitis are clinically silent. In less than half of the cases was diarrhea present. (2) The stool usually contains cysts, trophozoites, or both. (Stool examinations were done on 55 patients with positive results in 40.) (3) The disease usually occurs in men. (There were 69 men and 5 women in this series.) It rarely occurs in patients under twenty years of age. (4) Trauma seems to activate a latent infection of the liver. (5) Instead of being chronic, the process was an acute one in 78 per cent of the cases. (6) In 33 per cent of the cases abdominal features were absent or minimal and the clinical phenomena were pulmonary because the abscess was located in the dome of the liver. (7) Hepatomegaly is a constant feature. Even with dome abscess there is often downward dis-

largement. (8) Roentgen study will reveal the phrenic or transphrenic effects when the abscess is superior. The most characteristic changes are anterior and medial in the lower right chest. Elevation and immobility of the diaphragm are the earliest findings. Pneumonitis or pleuritis alone or in combination occurs as the lesion progresses. (9) Night sweats are frequent in the chronic cases and in the acute cases, chills are outstanding.

Treatment. The primary basis for cure is emetine in sufficient dosage. The author gives 1 grain daily for six doses, then a rest for six days after which the course is repeated as necessary.

Results of Treatment. (1) Nineteen patients (approximately 26 per cent) were cured by means of treatment with emetine alone. (2) Eighteen patients (approximately 24 per cent) were cured by means of emetine together with aspiration. (3) Surgery was done in 26 cases, 14 of which were adequately treated with emetine. The mortality was 53 per cent. This means that 12 cases were cured by surgery, some with and some without emetine.—*Mary Frances Vastine.*

SHEPARD, V. DUNCAN; WALTERS, WALTMAN, and DOCKERTY, MALCOLM B. Benign neoplasm of the gallbladder. *Arch. Surg.*, July, 1942, *45*, 1–18.

Benign tumors of the gallbladder are so rare that very few of them are seen even in a large surgical practice. Therefore, the authors have studied 150 cases seen at the Mayo Clinic from January 1, 1916, to December 31, 1938. They include 45 cases of polyps, 103 of adenomyoma and 2 of fibroma. All of these tumors occur in middle life, the most frequent incidence being in the fifties. Polyps and adenomyoma each occur in approximately 1 out of 100 gallbladders removed on operation.

These benign tumors are not found in gallbladders that are otherwise normal. In all these cases there were some signs of chronic inflammation and there were stones in more than half of them. The inflammation and the irritation caused by the stones are probably factors in causing the tumors. There is evidence that in some cases they may be due to displaced remnants of embryonic tissue.

The clinical symptoms of these tumors are not characteristic. The symptoms and physical findings are those of the accompanying cholecystitis or cholelithiasis. The only means of making a diagnosis before operation is by cholecystography. There is a clear defect in the shadow of the dye-filled gallbladder that is more radiolucent than that of the most radiolucent non-opaque stone.

These tumors rarely undergo malignant degeneration. Such changes were seen in only 2 of the cases of polyps and in none of the cases of adenomyoma or fibroma. But intramural abscess or intramural stone may occur in adenomyoma. Because of the possibility of these changes and of malignant degeneration and the frequency of inflammation and gallstones all gallbladders containing benign tumors should be removed.—*Audrey G. Morgan.*

PEARSE, HERMAN E. Whipple's disease. (Editorial) *Surg., Gynec. & Obst.*, Aug., 1942, *75*, 268–269.

In 1907 Whipple described a disease which was "characterized anatomically by deposits of fat and fatty acids in the intestinal and mesenteric lymphatic tissues," and which he termed intestinal lipodystrophy. This disorder caused loss of weight to the point of emaciation; loss of strength; microcytic anemia; recurrent arthritis; doughy swelling of the abdomen associated with tenderness and gaseous distention; abdominal pain and fatty diarrhea with an average of three or four stools a day. They were light or clay colored and consisted of over 50 per cent fat and fatty acids by weight. There was no interference with fat splitting for the fat in the stool was almost completely composed of split fat and soaps in crystalline form.

The disease is rare. The symptoms together with roentgen changes in the intestinal pattern may lead to a diagnosis of partial intestinal obstruction. It has been found that with adequate dosage of bile salts the symptoms disappear, appetite and strength return, weight is gained, vitamin A absorption improves and the fat content of the stool approaches normal.—*Mary Frances Vastine.*

JOHNSTON, CHARLES G. "Gas pains?" (Editorial) *Surg., Gynec. & Obst.*, Nov., 1942, *75*, 668–669.

The early recognition of ileus, whether occlusive or non-occlusive in type has long been recognized as being of importance in the control of mortality from this condition. It is to be remembered that it is not rare for patients with bowel obstruction to have some diarrhea, and

passage of gas or feces is not uncommon. There is but one laboratory finding of importance in the diagnosis of ileus and that consists of a flat roentgenogram of the abdomen. This is a simple painless laboratory procedure which yields rapid and definite data regarding the presence of gas in the small bowel long before abdominal distention is evident.—*Mary Frances Vastine.*

LEVITIN, JOSEPH, and TRAUNER, LAWRENCE M. A roentgenological study of the postoperative abdomen. *Surg., Gynec. & Obst.*, Oct., 1942, *75*, 510–514.

Distention frequently develops in twenty-four to ninety-six hours following abdominal surgery. A certain percentage of these cases are of a mechanical obstructive nature and not a paralytic ileus brought on by stimulation of the sympathetic nerves. These two conditions can be differentiated by a simple roentgen examination of the abdomen. The differential factors include: (1) a continuous distention to the point of obstruction in mechanical obstruction; scattered loops of distended small and large bowel in paralytic ileus; (2) loops few and large in mechanical obstruction; loops many and small in paralytic ileus; (3) dynamic appearance of bowel in mechanical obstruction; adynamic appearance of bowel in paralytic ileus.

This study is based on 107 cases from three sources of material. The conclusions reached from a study of these 3 groups are as follows:

1. Group 1. Unselected cases studied after operation.
   a. Distended bowel of a paralytic nature followed abdominal surgery in 75 per cent of cases.
   b. The roentgen findings of distention usually appeared on the first postoperative day and lasted three days.
   c. The type of anesthesia used seemed to play no part.
   d. Mechanical small bowel distention can exist without symptoms.
2. Group 2. Cases entering the hospital with abdominal distention and suspected bowel obstruction.
   a. Causes of mechanical obstruction in 29 cases included: adhesions 5, carcinoma of colon 5, volvulus 4, carcinoma implants 3, appendiceal abscesses 3, internal hernia 3, ileocolic intussusception 2, intussusception of Meckel's diverticulum 1, gallstone impacted in terminal ileum 1, perforated bladder 1, mesenteric adenitis 1.
   b. Causes of paralytic ileus in 6 cases included: acute appendicitis 2, perforated duodenal ulcer 1, inguinal hernia 1, bleeding follicular cyst 1, mesenteric thrombosis 1.
   c. The absence of roentgen findings of distention does not rule out a severe bowel pathological lesion. This is especially true in cases of strangulated bowel.
3. Group 3. Cases of postoperative distention not included in Group 1.
   a. All cases of the abdomen examined roentgenologically will not fit into one of the three groups of mechanical obstruction, adynamic ileus, or no distention. For instance, there may be a combination of local ileus and mechanical block.
   b. The use of the Miller-Abbott tube is not advised for the patient entering the hospital with a low intestinal obstruction. It is recommended for use in the patient with postoperative distention.—*Mary Frances Vastine.*

GYNECOLOGY AND OBSTETRICS

HASTINGS, W. H. Pelvimetry simplified. *Brit. J. Radiol.*, April, 1942, *15*, 114–121.

In measuring the diameters of the female pelvis by roentgenometric methods, the first essential is to determine the height of the diameter to be measured above the film at the time of exposure. Once this is known the question is one of simple proportion. A method is described in which two exposures are made on the same film with a double shift of the tube of 1½ inches to either side of the midline, the total shift being exactly 3 inches. The patient need not hold her breath but must not move until the second exposure is completed. The focus-film distance is 30 inches.

Based on this focus-film distance and shadow shift, a table, which is reproduced, has been worked out, showing the heights of the various points corresponding to the different measurements of their shadow shifts. With this and a simple chart made out on squared graph paper of a scale of 1 inch to the square, subdivided vertically and horizontally into tenths of an inch, the true diameter can be read off immedi-

ately after measuring the shadow shift and the distance between the bisecting points of the shadow shifts of the two ends of the diameter and after referring to the table of heights. The chart is also reproduced. The computation can be made accurately in a few minutes for all diameters and angles of the pelvis without any arithmetical calculations.—*Audrey G. Morgan.*

ROSENBERG, MAXIMILIAN, and SCHENCK, SAMUEL B. Gestation fourteen years after radium induced amenorrhea. *Am. J. Obst. & Gynec.*, June, 1942, *43*, 1033–1035.

A white woman, aged forty-seven, was admitted with a history of backaches for six months and amenorrhea for fourteen years following radium therapy. She appeared acutely ill. She had received 1,200 mg-hr. of radium when she was thirty-three years of age because of menorrhagia and metrorrhagia. (Several months prior to this, a therapeutic abortion had been performed for pernicious vomiting of pregnancy.) At the time of the present admission a diagnosis of submucous fibroid was made as the uterus was found to be the size of a three months pregnancy. A supracervical hysterectomy was done and the pathologic diagnosis was that of a gravid uterus with placental tissue.—*Mary Frances Vastine.*

HIRSCH, J. B., and HIRSCH, J. B., JR. Normal infant and a sympus monster with a short cord in a twin pregnancy. *Am. J. Obst. & Gynec.*, May, 1943, *45*, 889–891.

There are many records of sympus monsters in the literature but this case is unusual in the occurrence of such a monster in a twin pregnancy.

Roentgen findings on the monster: (1) thirteen ribs on left side; 12 ribs on the right; (2) angle of mandible absent on right side; (3) impression of complete spina bifida although the spinous processes were normal in number; (4) coccyx anterior to femur; (5) pelvis consisting of a right and left os ilium which were asymmetrical with a heart-shaped bone between them; (6) no real hip joint but two femurs were somehow articulated on each side between the heart-shaped bone and the ilia; (7) fusion of lower extremities with two femurs, two tibias, and one fibula. (8) some separation distinguishable between the soft parts of the femurs but not between the tibias; (9) only one foot which projected at a right angle from the distal end of the left tibia. One tarsal bone, six metatarsals and six phalanges were present.—*Mary Frances Vastine.*

RICE, MAURICE, PEARSON, BJARNE, and TREADWELL, W. B. Malignant lymphangioma of the ovary. *Am. J. Obst. & Gynec.*, May, 1943, *45*, 884–888.

A case of an apparently morphologically benign lymphangioma of the ovary is presented in which a resection was done. Six months later, a local recurrence with peritoneal dissemination and hepatic metastasis had taken place and a new growth in the opposite ovary was present. A marked morphologic difference was evident between the primary and disseminated neoplasm consisting of proliferation of the endothelium and changes in the individual cells. However, because of the marked necrosis of the primary together with the marked hepatic necrosis present at autopsy, the authors feel that this tumor was malignant at its inception in spite of the regular benign morphology it presented.—*Mary Frances Vastine.*

TAUSSIG, FRED J. Iliac lymphadenectomy for group II cancer of the cervix. *Am. J. Obst. & Gynec.*, May, 1943, *45*, 733–748.

From 1930 to October, 1942, the author and his associates have done 175 iliac lymphadenectomies. The author is convinced that in group II cancers of the cervix as an addition to the thorough irradiation of the primary tumor, iliac lymphadenectomy is of definite value.

The preferred plan of procedure is as follows: (1) A course of roentgen therapy four times a week for about four or five weeks is given through six portals, two anterior, two posterior and two perineal. A tumor dose of 2,500 to 3,000 r is delivered. The midline area where the operative incision is to be made is protected during treatment. (2) Operation (i.e., inguinal lymphadenectomy) is performed two weeks following termination of the roentgen treatment. (3) Radium treatment is given eleven to fourteen days following the operation. A dosage of 5,000 to 6,000 mg-hr. is delivered. The application is made from five to six points (corpus, cervical canal, against the cervix, in the vaginal fornices), using a total of 125 to 135 mg. in 0.5 mm. platinum for forty to forty-five hours.

The author summarizes his work as follows: (1) One hundred and seventy-five cases of group II cervical cancer were subjected to iliac

gland removal with irradiation of the primary tumor. The operative mortality was 1.7 per cent with a mortality of 0.7 per cent in the last 140 cases.

(2) The five year survival rate of 70 cases operated on previous to October, 1937, was 38.6 per cent. This is to be compared to a five year survival of 22.9 per cent in 118 similar cases treated by irradiation alone.

(3) Cancer was found to be present in the lymph glands in 26.8 per cent of the 175 cases operated upon. Even in those cases with demonstrable metastases, a five year salvage of 21 per cent was obtained.—*Mary Frances Vastine.*

MEZER, JACOB. Metaplasia and carcinoma in cervical polyps. *Surg., Gynec. & Obst.*, Aug., 1942, *75*, 239–244.

A cervical polyp is simply a localized heaping up of normal cervical tissue. Cervcial polyps appear most frequently in the fifth decade and in parous women.

One thousand six hundred and thirty-six polyps were studied at the Free Hospital for Women in Brookline, Mass. It was concluded that: (1) cervical polyps are frequently found in gynecological patients; (2) a third of the polyps studied were subject to squamous metaplasia, or "epidermization," which is neither a precancerous nor a malignant process; (3) 5 cases of carcinoma which arose in cervical polyps are cited among the 1,636 polyps studied (an incidence of 0.03 per cent); (4) cervical polyps should be removed and their bases cauterized; (5) in cases of abnormal bleeding, other causes should be ruled out by means of careful pelvic examination before the cervical polyp is accepted as the only cause of the bleeding.—*Mary Frances Vastine.*

GLASS, MORRIS, and GOLDSMITH, JOSEPH W., JR. Fibroma of the ovary associated with ascites and hydrothorax (Meigs' syndrome). *Am. J. Obst. & Gynec.*, June, 1942, *43*, 1048–1054.

In 1923, Owen and Horn described the association of an ovarian fibroma with hydrothorax and ascites. Meigs and Cass in 1937 called attention to this clinical entity and they emphasized the fact that such cases could be cured by removing the ovarian tumor in spite of the grave appearance of the patient.

Eighteen cases of Meigs' syndrome have been previously reported. The authors report this, the nineteenth case, in a woman seventy-three years of age. The patient was hospitalized with symptoms which caused a diagnosis of cardiac decompensation to be made. Roentgenograms of the chest revealed a pleural effusion on the right extending to the sixth rib in the axillary line. Bomze and Kirschbaum point out that many of these patients have been hospitalized for symptoms suggestive of heart failure. The reason for the fluid accumulation is not known. It has the physical and chemical characteristics of a transudate. The pleural fluid is more frequently found in the right chest cavity. Following removal of the ovarian tumor, the fluid spontaneously disappears although it rapidly re-accumulates following thoracentesis or paracentesis before removal of the neoplasm.—*Mary Frances Vastine.*

FOLSOM, ALFRED I., and O'BRIEN, HAROLD A. The female obstructing prostate. *J. Am. M. Ass.*, Feb. 20, 1943, *121*, 573–580.

In 1931, Folsom first emphasized the clinical importance of the group of glands surrounding the posterior part of the female urethra. He felt then, and the writers feel now, that an infection harbored through the years in these glands is probably the cause of the bizarre pathologic picture seen in this portion of the female urethra. Prostatism is a clinical entity that is seen much more frequently in women than is recognized and properly treated.

Clinically these cases present, in addition to a bladder irritation, some degree of difficulty in voiding. This varies from simply a sense of obstruction and unfinished business on through varying degrees of obstruction to complete retention. A plain cystogram, either air or sodium iodide being used, will frequently reveal a filling defect in the region of the internal orifice similar to the defects seen in men with prostatic hypertrophy. On cystoscopic examination one may find a normal bladder wall or a grossly trabeculated wall with cellules and even diverticula and calculi. Not infrequently the bar of Stilwell may be felt by palpating the urethra through the vagina with the cystoscopic sheath in place. The majority of the cases show a fibromuscular hypertrophy with fibrous hyperplasia and varying grades of inflammatory reaction. In some cases actual gland structures have been reported, and these seem to be identical with similar lesions in the male.

The treatment of this condition (trans-

urethral resection) is described, and 7 case histories are given.—*S. G. Henderson.*

EHRLICH, H. E., and HOROWITZ, E. A. Benign papillary epithelioma of the vulva. *Am. J. Obst. & Gynec.*, May, 1943, *45*, 879–884.

A case of benign papillary epithelioma is described and 4 similar authentic cases are collected from the literature. The characteristics of this tumor include the following: (1) It occurs as a single, round or ovoid mass whose surface is papillary and nonulcerated. (2) It is attached to the labia or mons veneris by a short pedicle or it may be sessile. (3) Unlike condyloma acuminatum, it occurs as a single growth, reaches appreciable size and is not associated with a foul odor. (4) Histologically, it differs from the venereal wart by the presence of intense keratosis and parakeratosis, absence of edema of the epiderm, but little stigma of inflammation, and a connective tissue stroma that is not congested. (5) The benign epithelioma of the vulva does not recur after operative removal. (6) Carcinoma-like condylomas of the penis have been described. These have been regarded as benign and are characterized by a downward infiltration of the epithelium. Such a picture is strikingly absent in benign papillary epithelioma of the vulva.—*Mary Frances Vastine.*

GRAVES, SIDNEY C., and MEZER, JACOB. Malignancy of the vulva. *Am. J. Obst. & Gynec.*, June, 1942, *43*, 1016–1021.

At the Free Hospital for Women, in Boston, between January, 1890, and January, 1941, 66 cases of cancer of the vulva were seen. In the same interval there were 1,668 cases of carcinoma of the cervix, 475 of the body of the uterus, 179 of the ovary, and 562 of the breast.

(1) Cancer of the vulva is an uncommon disease occurring in 0.2 per cent of the patients at the Free Hospital for Women. (2) It is a disease primarily of women past the age of fifty. (3) It is usually associated with leukoplakia and kraurosis of the vulva. Chronic bartholinitis, or venereal warts, may be predisposing factors. (4) The usual pathologic picture is that of epidermoid carcinoma. Adenocarcinoma or sarcoma is occasionally the type of malignancy found. (5) For all patients entering this hospital the five year salvage is 27.7 per cent. The operative mortality is 5.4 per cent. (6) The treatment of choice is a radical vulvectomy plus a bilateral groin dissection. (7) Irradiation of the vulva should be used only as a last resort.—*Mary Frances Vastine.*

GENITOURINARY SYSTEM

PIERSON, L. E., and HONKE, E. M. Respiration pyelography in the diagnosis of perinephric abscess. *J. Urol.*, May, 1942, *47*, 580–581.

Bacon has found that the average cranio-caudad excursion of each kidney is 2.75 cm. and the mesiolateral excursion 0.33 cm. On this basis, the value of respiration pyelography has its foundation.

The author advises having the patient employ abdominal breathing, using great care that no change in body position occurs. Bilateral pyelograms are essential for comparison. A survey film is first exposed, the medium injected and the catheters plugged. The third film is exposed twice, at the end of deep inspiration and at the end of deep expiration. Seventy-five per cent of the intensity of a single exposure is employed.

In the presence of a normal kidney a double pyeloureterogram results. In the presence of a perinephric abscess only a single pyeloureterogram results on the affected side.

Other factors which may confuse the diagnosis by affecting kidney mobility are inflammatory processes of the perirenal fascia, perirenal adhesions, gallbladder disease, lumbar scoliosis, phrenic nerve section and liver infections.

An illustrative film is reproduced.—*R. M. Harvey.*

PRINCE, C. L. Primary angio-endothelioma of the kidney; report of a case and a brief review. *J. Urol.*, June, 1942, *47*, 787–792.

Angio-endothelioma of the kidney is an extreme rarity. Prince reports a case in a white male, aged fifty-one, who had had pain in the left flank for two weeks. Intravenous urography showed a non-visualized left kidney. A retrograde pyelogram showed marked hydronephrosis of the left kidney.

A nephrectomy was performed when no function was present in the left kidney after a three week interval. A large tumor mass was present on the anterior surface of the kidney pelvis. Histopathological examination revealed a very malignant angio-endothelioma. There was almost no recognizable kidney substance. Postoperative radiation treatment was given with 6,300 r over the kidney area, through three

portals. The factors are not stated. Since the tumor was well encapsulated, showed no local extension and no general metastasis, the author feels the prognosis is hopeful.

The accepted treatment for this condition is operative removal followed by postoperative deep roentgen therapy.—*R. M. Harvey.*

SARNOFF, S. J. The incidence of intrarenal kidney pelvis in essential hypertension. *J. Urol.*, June, 1942, *47*, 769–775.

Ravich has noted that the kidney pelvis in the newborn is enclosed within the renal parenchyma and gradually assumes an extrarenal position. In some individuals the intrarenal type of pelvis may persist into adult life. It seems likely that such an intrarenal type of pelvis might not have the freedom of expansion in the presence of an obstructive lesion of the urinary tract. That is, in expansion, pressure would be produced on the renal vessels resulting in renal ischemia and hypertension.

In order to investigate this problem the author examined the excretory urograms of 50 patients from the Hypertension Clinic of the Johns Hopkins Hospital. One hundred urograms from routine check-ups on patients with no demonstrable renal pathema or hypertension were used as controls. He found that errors in the diagnosis of intrarenal pelvis might occur due to rotation of the kidney and due to the presence of borderline cases.

The results of this experiment showed that only 40 per cent of hypertensive cases had an intrarenal type of pelvis whereas 30 per cent of the control cases had this anomaly. Therefore, intrarenal type of pelvis would not seem to be a factor in the production of hypertension—*R. M. Harvey.*

KRETSCHMER, HERMAN L. The diagnosis and treatment of tuberculosis of the kidney. *Surg., Gynec. & Obst.*, Dec., 1942, 75, 704–711.

It is believed today that the cause of renal tuberculosis is infection by the hematogenous route and the fixation of tubercle bacilli in the tissue. The primary infection is usually in the respiratory tract.

This study is based on 95 cases of tuberculosis of the kidney which the writer has seen since his last publication on this subject in 1936. The findings and conclusions are as follows:

Sex. Practically no difference in the sex incidence.

Age. The largest number of cases occurred in the third decade.

Sides. Bilateral involvement occurs relatively frequently. In this series 23.1 per cent were bilateral. The right side was involved in 44.2 per cent and the left side in 32.6 per cent.

The Presence of Pulmonary Involvement. One-half of the cases showed evidence of pulmonary tuberculosis, either active or healed.

Other Tuberculous Lesions. Thirty-nine per cent of the patients had evidence of tuberculosis in other parts of the body. A large number had genital tuberculosis while those with glandular or bone tuberculosis were few in number.

Diagnosis. History of tuberculosis, pus and tubercle bacilli in the urine establish the diagnosis of renal tuberculosis. Stone associated with tuberculosis is uncommon. Areas of calcification are of diagnostic importance but they are not often seen.

1. *Intravenous urography.* An intravenous pyelogram reported as normal does not rule out a tuberculous kidney. Thus Emmett found tubercle bacilli in the urine of 26 per cent of his patients in whom the intravenous pyelogram was reported as normal.

2. *Cystoscopic examination and ureteral catheterization.* Repeated instrumentation should be kept at a minimum. Early, the bladder may be normal. Late, it may be extensively involved with limitation in capacity. Care must be taken not to infect the normal ureter.

3. *Demonstration of tubercle bacilli in urine.* Tubercle bacilli were found in 85.3 per cent of the cases; in 14.75 per cent, the diagnosis was made by cystoscopy and by pyelograms.

4. *Retrograde pyelography.* This aid to diagnosis is resorted to when the intravenous urogram has been reported as normal and the examination of urine from the supposedly normal kidney discloses the presence of pus cells. Furthermore, in cases in which the tuberculous lesion is very small and an intravenous urogram is inadequate to show its presence, a retrograde pyelogram should be of considerable value.

Treatment. Once the diagnosis of renal tuberculosis has been established the treatment is nephrectomy. Renal tuberculosis as seen by the clinician does not heal. When the involvement is bilateral, the author refrains from doing a nephrectomy on the more involved kidney, unless sepsis supervenes.—*Mary Frances Fosnine.*

CREEVY, C. D. An example of apparent healing of bilateral minimal renal tuberculosis. *J. Urol.*, May, 1942, *47*, 614–618.

It is now becoming evident from case reports in the literature that destructive renal tuberculosis may heal spontaneously. True healing with disappearance of tubercle bacilli and replacement by fibrous tissue must be distinguished from pseudo healing in which the tuberculous infection is isolated by scar tissue from the lower urinary tract, permitting clearing of the urine and healing of bladder lesions. In the latter condition viable organisms persist and potential activation is a constant threat.

Renal tuberculosis is a chronic systemic disease and haste in performing a nephrectomy on an infected kidney is not warranted. In bilateral destructive renal tuberculosis conservative sanatorium care is indicated. Also in unilateral destructive tuberculosis, in the presence of an active extra-urogenital lesion, conservative care is justified until the extra-urogenital focus can be brought under control.

Close expert supervision is required during this conservative regimen.

The author reports a case of bilateral pulmonary tuberculosis in a woman aged twenty-five, who had been under treatment for two years before urinary symptoms developed and bilateral minimal destructive renal tuberculosis could be demonstrated by pelography.

The diagnosis was confirmed by guinea pig inoculations with urine from each kidney. Bed rest, artificial pneumothorax, and general hygienic measures resulted in arrest of the pulmonary lesions, disappearance of urinary symptoms, clearing of the urine and urographic evidence of calcium deposition in the renal lesion of the right side.

Eleven years have elapsed since the onset with no evidence of reactivation. While the author does not advocate conservative measures in destructive unilateral renal tuberculosis without an extra-urogenital lesion, he does wish to point out that such lesions may heal under sanatorium care.—*R. M. Harvey.*

LANGWORTHY, H. T., and DREXLER, L. S. Carcinoma in crossed renal ectopia. *J. Urol.*, June, 1942, *47*, 776–783.

In unilateral fusion of the kidneys and unilateral double kidney both ureters empty into the same side of the bladder. In crossed renal ectopia both ureters empty into their normal openings into the bladder. Only 20 cases of crossed renal ectopia with non-fusion of the kidney have been reported. Carcinoma has never been reported previously in crossed renal ectopia.

The authors report 2 cases of adenocarcinoma in crossed ectopic kidneys. The diagnosis was made in each instance at operation. The preoperative diagnosis in the first case, made on the retrograde pyelogram, was crossed renal ectopia with hypoplasia and infection of the ectopic kidney. The second patient did not have preoperative urographic studies made.—*R. M. Harvey.*

SPARKS, A. J. Wilms' tumor in a 63 year old male. *J. Urol.*, May, 1942, *47*, 642–647.

Wilms' tumor is a very malignant tumor of infancy and early childhood and its occurrence in adults is extremely rare. Twenty-six cases of Wilms' tumor have been reported in adults. The author reports an additional case in a sixty-three year old male who complained of right lumbar pain and hematuria of eight weeks' duration. A retrograde pyelogram showed enlargement of the right kidney with dilated calyces and a large filling defect in the right renal pelvis.

At operation a tumor was found in the lower pole which proved to be embryonal carcinoma. The patient died of postoperative pneumonia.

The accepted treatment for Wilms' tumor in children is nephrectomy with pre- and postoperative irradiation. However, in adults it is practically impossible to make the diagnosis preoperatively. The author suggests that in the presence of a large tumor mass, toxemia, and weakness out of proportion to the apparent duration of the lesion, all signs suggestive of a rapidly growing tumor, it might be advisable to delay operation and give preoperative irradiation.—*R. M. Harvey.*

MISCELLANEOUS

STONE, ROBERT S., and LARKIN, JOHN C., JR. The treatment of cancer with fast neutrons. *Radiology*, Nov., 1942, *39*, 608–620.

Fast neutrons are nuclear particles that have no electrical charge. These particles when accelerated deliver a new kind of radiation. The cyclotron at the Crocker Radiation Laboratory, University of California, supplies beams of fast neutrons of great enough intensity for biological and therapeutic purposes. It produces deu-

terons with an energy of 16 mev. (million electron volts). The physical factors involved are different from those used in measuring roentgen rays so an arbitrary unit called the neutron unit, or n, has been adopted. One neutron is practically equivalent to 6 r and the 110 n required to produce a minimum threshold reaction on the skin is roughly equivalent to 650 to 700 r measured on the skin.

These fast neutrons have been used in the treatment of 120 cases of cancer of various organs and regions of the body and of varying degrees of intensity, though all were inoperable and incapable of cure by roentgen treatment, as experimentation was not considered justifiable in operable cases.

Tables are given showing the details of the skin reactions of the neck and face. A table is also given showing the effect of the irradiation on the cancers. At the end of the treatment the lesions had completely regressed clinically in 20 of the 120 patients. Three months after treatment 26 patients were clinically free of cancer in the affected areas. In 57 patients there was partial regression and in 43 no regression at all. It should be remembered that some of these patients were in such an advanced state of disease that they should probably not have been treated. There were severe general reactions in some of the cases and experimental work is still going on with regard to the best technique of administration. In general, the methods are the same as those used in roentgen therapy but it is possible that greater good will be done by more massive doses of neutrons. Primary tumors seem not to have responded to the neutrons as well as metastases. It would seem from experiment so far that the total dose should be given in a much shorter time than it was at first.

While the results so far do not seem very encouraging it is believed they justify further experiment with the use of fast neutrons and even their use in earlier stages of the disease. —*Audrey G. Morgan.*

Goldfeder, Anna. Relation between radiation effects and cell viability as indicated by induced resistance to transplanted tumors. *Radiology*, Oct., 1942, *39*, 426–431.

Experiments on mice are described in which fragments of mouse sarcoma 180 were irradiated and then implanted. A study is made on the effect of previous irradiation on the taking of the implants and the creation in the animals of resistance to the taking of later implants of fresh tumor material. Tables are given showing the details of the results.

It was found that if sarcoma 180 was irradiated with a dose of 60,000 roentgens not only was the proliferating power of the sarcoma destroyed but the active principle that induced resistance in the mice to subsequent implantations of tumor tissue of the same strain was also destroyed. But when much smaller doses were used, such as 4,000 to 5,000 r, the implants did not produce any detectable tumor but they did make the mice refractory to later implants of the same strain.

The author does not intend to advocate the use of tumor implants properly attenuated by irradiation in the treatment of human tumors, but she does believe that these results suggest further research on the possibilities of such a method of treatment.—*Audrey C. Morgan.*

Marshak, Alfred. Effects of x-rays and neutrons on mouse lymphoma chromosomes in different stages of the nuclear cycle. *Radiology*, Nov., 1942, *39*, 621–628.

Experiments in the irradiation of mouse lymphoma chromosomes with roentgen rays and fast neutrons are described. They show that the chromosomes of the mouse lymphoma are sensitive to irradiation not only in the beginning of of the prophase but that they are also even more sensitive during the resting stage.

Details of the technique of the experiments are given and graphs and tables showing the results. The curves show that the percentage of chromosomes that remain normal at eight, twelve and twenty-four hours after treatment with either roentgen rays or neutrons is a negative exponential function of the dose. At eighteen hours the curves are composite one part showing the slopes of the twenty-four hour curves, the other at lower doses the slopes of the twelve hour curves.

The ratio (n/x) of the neutron curves to the roentgen-ray curves shows the relative efficiency of treatment of these lymphomas with neutrons and roentgen rays. It is stated here that the n unit is equivalent to about 2 or 2.5 roentgens. But independently of the units used to measure dosage neutrons cause greater damage to chromosomes than roentgen rays. With the root tip of the horse bean (*Vicia faba*) the ratio n/x varies from 6.6 to 15; that is, neutrons are more than twice as effective in causing damage in the resting stage; with the mouse

lymphoma it rises from 5.8 to 8.8 in the resting stage.

As neutrons cause relatively greater damage to the chromosomes during the resting stage than roentgen rays it is to be expected that in neutron treatment the effects will be less dependent on the rate of mitosis and that therefore tumors that are resistant to roentgen treatment may yield to treatment with neutrons.—*Audrey G. Morgan.*

Silverstone, Sidney M., and Wolf, Bernard S. A study of the limiting diaphragm method of collimation. *Radiology*, Sept., 1942, *39*, 314–319.

The term limiting diaphragm means a diaphragm placed close to the target for the purpose of limiting the roentgen-ray beam to a specified field at a specified target-surface distance. Roentgenograms and graphs are given illustrating collimation by the limiting diaphragm method. The disadvantages of the method are that the distribution of the dose over the irradiated field is non-uniform and asymmetrical and there is a marked decrease of the surface and depth doses at the periphery of the irradiated volume as compared with the doses in the central axis at the same level. There are also variations in output for different sizes of field.

The authors find it preferable to eliminate the diaphragm entirely and collimate the roentgen-ray beam, using only a cone with lead-lined walls. This gives a uniform beam with no penumbra and the shadow is homogeneous and sharply defined for the specified field size. The maximum peripheral doses are obtained on the surface and in the depths of the phantom. The output for any target-surface distance is independent of the size of the field and slightly greater than if a limiting diaphragm had been used.

Commercial cones from three manufacturers of roentgenologic equipment were studied. Two had limiting diaphragms and showed all the defects described above. In the other, lead-lined walls were used without a limiting diaphragm and the results were satisfactory.—*Audrey G. Morgan.*

Jenkins, James A., and McGeorge, Murray. Control by radium for gastric acidity. *Arch. Int. Med.*, Nov., 1942, *70*, 714–721.

The authors describe experiments in the use of radium for the reduction of hyperacidity in gastric and duodenal ulcer. Tubes containing two 25 mg. needles or four 10 mg. needles of radium were fastened in the end of a stout rubber tube which was swallowed. The end of the tube was surrounded by a rubber bag full of water which prevented the needles from passing into the duodenum was kept them away from the stomach wall. Treatment was given for four or five hours daily up to a total dose of 2,000 to 2,500 mg-hr.

To determine the secretory activity of the gastric mucosa they used not only the gruel test meal but also the subcutaneous injection of histaminic acid phosphate which can be standardized. Graphs are given showing the effect of the radium treatment on gastric secretion.

Among the 14 patients treated, most of whom had duodenal ulcer, acidity was reduced in 13. It is not yet known whether this reduction will be permanent but there are some indications that it will later rise to some extent.

Among the 13 patients in whom acidity was reduced 7 were completely relieved of clinical symptoms without further alkali treatment and 5 were improved. There was no improvement in the other case but later operation showed that the ulcer had penetrated the pancreas, which explained the failure.—*Audrey G. Morgan.*

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THE AMERICAN JOURNAL OF ROENTGENOLOGY AND RADIUM THERAPY

VOL. 51 JUNE, 1944 NO. 6

ACTINOMYCOSIS OF THE VERTEBRAE

By MORTIMER LUBERT, M.D.

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ACTINOMYCOSIS of the vertebrae is a rare manifestation of a relatively rare disease. Yet this unusual complication presents such a characteristic roentgen appearance when it does occur that it is strange to find little mention of it in roentgenologic textbooks, and only scattered mention of it in roentgenologic periodicals, the latter consisting of brief summaries of articles originally presented elsewhere.

In the past year, 2 cases of actinomycosis with vertebral involvement have occurred at City Hospital and a less recent case has been found at the University Hospitals of Cleveland, all of which present lesions well demonstrated on the roentgenogram. It is the purpose of this paper to report these 3 cases with postmortem as well as roentgen findings.

INCIDENCE

In 1923 Sanford[11] published a series of 678 cases of actinomycosis, only 4 of which had rib or vertebral involvement. At approximately the same time Parker[10] presented 9 cases of vertebral actinomycosis which he collected from the literature. By 1927 Simpson and McIntosh[13] were able to collect from the world literature 28 cases of vertebral actinomycosis, 13 of which were in the American literature. To this group they added 4 of their own cases. Snoke[15] added a case in 1928 and Ward[17] another in 1931. In 1933 Tabb and Tucker[16] again reviewed the subject and added another case report. Their presentation has the only illustrations of the roentgen manifestations of the disease in recent American roentgenologic periodicals. In 1935 Meyer and Gall[8] reviewed the literature completely and collected 47 cases of vertebral actinomycosis. They present an excellent study of the subject, but without illustrative roentgenograms. Other articles presenting single cases have occurred subsequently; the one presented by Dixon[4] resembles one of the cases presented here in that collapse of a vertebra occurred. This is a rare phenomenon. Since many of the cases are usually called Pott's disease before autopsy it may be that with greater awareness of its existence the diagnosis of vertebral actinomycosis will be made more frequently.

ETIOLOGY

The precise etiology of actinomycosis has been the subject of much debate, but in recent years most of the argument has been resolved mainly by the researches of Naeslund[9] and the popularization by Cope[1,2] It was generally agreed that actinomyces caused the disease. However, the pathogenicity of the various varieties, the cultural characteristics of the organisms in

covered in human lesions, the life cycle of the various types with the possibility of one type reverting to another in a different phase of development, and even the nomenclature were at times parts of an active controversy.

The present conception of actinomycosis is that it is caused in a great preponderance of cases by an anaerobic organism which normally inhabits many human mouths. On rare occasions an aerobic organism which has its origin outside the body may be responsible. The organisms found on grasses and grains are aerobic, but are not believed to be pathogenic. The historical development of these concepts is interestingly presented by Cope.[1]

These beliefs are presented for more than academic interest. They are important for several reasons: first, the victim of the disease need not be a farm worker or one who has had contact with cattle; second, he need not have chewed grasses, straws, or other vegetable matter; and third, such incidents in the patient's history as a recent tonsillectomy, poor mouth hygiene, a recent tooth extraction or a human bite are as much or even more important in suggesting actinomycosis than the occupational and social history. Many recent articles[2,3,7] support these views. Nevertheless their appreciation is not widespread.

At the present time there has been a general tendency to abandon all the confusion in terminology with the acceptance of the general term, "actinomyces," for the entire group of organisms and "actinomycosis" for the disease. The term, "streptothrix," has largely been dropped except as a subtitle. However Lord[7] still believes it to be a useful designation for the rare aerobic form. He believes morphological and prognostic differences substantiate his point. Cope, on the other hand, is not impressed and believes unity in nomenclature is more valuable.

PATHOGENESIS AND PATHOLOGIC FINDINGS

The vertebrae are involved secondarily usually by direct extension but occasionally by blood stream metastasis. The organism sets up the primary focus by passing through the mucous membranes of some portion of the respiratory or gastrointestinal tract. Occasionally entrance may be made through the skin. Suppuration begins and its main feature is extensive burrowing with little respect for anatomical barriers. Thus the vertebrae can be reached from almost any primary site, but mainly from pulmonary and gastrointestinal foci.

When the infection reaches the vertebrae it is usually in the form of a paravertebral phlegmon and in the abdomen may resemble a tuberculous psoas abscess. The cortex of the vertebrae is first eroded and abscesses are set up beneath it. Osseous condensation takes place surrounding the abscesses at the center of which the organisms may be found. While all portions of the vertebra may be involved, including the processes and lamina as well as the bodies, the intervertebral discs are usually spared and angular deformity is rare.

Pathologically, the disease belongs to the group of chronic granulomas. However, there are several interesting features peculiar to it. In the early stages extreme vascularity is one of them. During surgical operation, bleeding is profuse and the alert surgeon[13,16] may suspect the presence of the disease at the time of operation because of it. When actinomycosis has been present for a longer period of time, much glistening, firm, connective tissue is laid down in the form of thick strands which surround small areas of suppuration at the center of which the ray fungus is found. Microscopically, there are present in the very vascular pyogenic granulation tissue many pale vacuolated cells which are not found in tuberculosis.[13] These cells may suggest the disease when the ray fungi cannot be found and more sections can then be taken and the responsible organism more closely searched for.

CLINICAL FINDINGS

The clinical findings in actinomycosis are those of any chronic suppurative process. Fever, loss of weight and appetite, pain, and weakness are part of the generalized picture.

There are usually many draining sinuses. Secondary infection may complicate the picture and conceal the presence of the ray fungus. Amyloid disease may result. The local symptoms will depend on the site of major involvement. Thus pulmonary actinomycosis may simulate tuberculosis or cancer in its clinical manifestations. Involvement of the gastrointestinal tract will simulate other granulomatous or neoplastic disease in this system. When the spine is affected back pain becomes an important feature. Neurological manifestations do occur, but are rare.

DIAGNOSIS

Recovery of the actinomyces in the discharges from the sinuses or their demonstration in histologic preparations are the only positive ways of making the diagnosis. Roentgen examination, however, may be helpful.

ROENTGENOGRAPHIC FINDINGS

The roentgen examination may aid in the diagnosis of actinomycosis before the vertebrae are involved especially where the lungs are the primary site. Kirklin and Hefke[6] have pointed out that the involvement of the ribs and sternum in association with parenchymal infiltration in the lungs, pleural thickening, and empyema more strongly indicates actinomycosis than any other process. The ribs and sternum may show destruction with or without the reaction of osteomyelitis or periostitis about them. If sinuses are present their connection with the destroyed rib need not be demonstrated. The laminagram will probably be of inestimable value in demonstrating lesions of the sternum.

When the vertebrae are involved the roentgen findings may be characteristic if not pathognomonic. Erosion of all portions of the vertebrae and adjacent ribs including the pedicles, spines, lamina and body is a feature. The body is usually affected in its cortical portion with periostitis as an early manifestation finally leading to erosion. The anterior surface of the body may have a saw-tooth appearance.[8] Reactive condensation takes place about destroyed areas and the vertebra involved may appear denser than its neighbors. The intervertebral disc is usually not involved. In no case is the uniform decalcification of one or more vertebrae found as is sometimes observed in tuberculosis.

Both tuberculosis and nonspecific osteomyelitis may on rare occasions simulate the appearance of actinomycosis of the vertebrae. However, involvement of the intervertebral disc with narrowing of the intervertebral space and finally collapse of the body of the vertebrae is the most common type of tuberculous involvement. Any deviation from this picture makes the roentgen diagnosis of tuberculosis difficult without supporting evidence from other sources. In nonspecific osteomyelitis biopsy and bacteriological examination must be resorted to, if the rest of the clinical picture is not conclusive. Even then difficulties may remain as actinomyces may not be present in a small biopsy specimen, and a prolonged and repeated search will be necessary before organisms are demonstrated.

CASE REPORTS

CASE 1. J. S., Negro, male, age [illegible], was admitted to the Surgical Service on June 26, 1941, with a mass 4 cm. in diameter on the right side of the neck and another just to the left of the sternum. Both masses were definitely fluctuant, but not hot or tender. A draining sinus was present over the tibia. Another sinus on his head had healed. He had lost 25 pounds in weight in the five months prior to admission. As an inmate of a prison farm, he had contact with cattle and had chewed grasses and straws.

Physical examination on admission revealed a temperature of 38.8° C., a pulse of 130 per minute, and respirations of 22 per minute. The head was held to the right. The mouth and throat appeared normal. The teeth were in good condition. Aside from the masses described, the draining sinus on the left tibia and signs of emaciation, the rest of the physical examination, including the neurological examination, was negative.

The laboratory findings showed the urine and blood to be normal. The Kline exclusion test was negative.

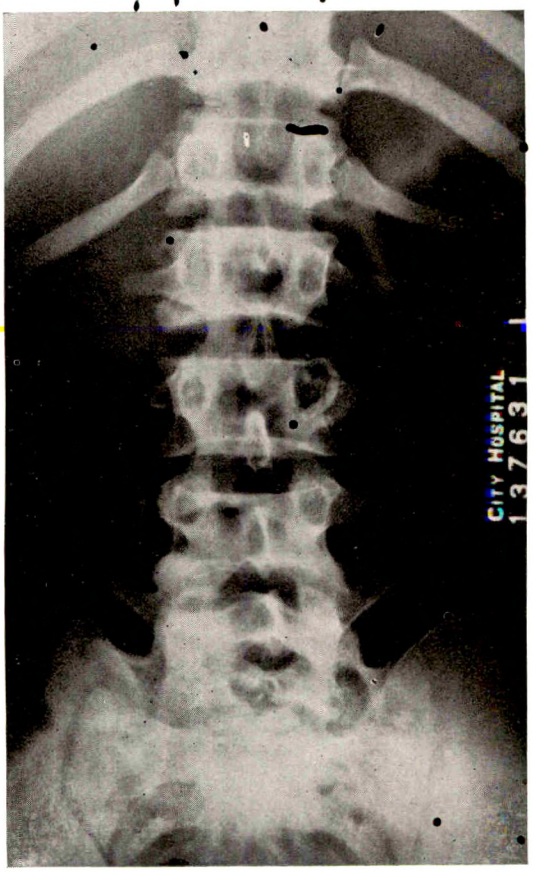


FIG. 1. Case 1. Anteroposterior view shows areas of destruction in the lumbar vertebrae.

Roentgen examinations revealed enlargement of the right hilum probably due to enlarged lymph nodes, but no definite evidence of pulmonary infiltration. There was an area of destruction in the right fourth rib posteriorly. The cervical spine appeared normal. There were areas of destruction in the left frontal bone. These findings were interpreted as osteomyelitis and mediastinal lymphadenitis of tuberculous origin. Roentgenograms of the vertebrae taken two months later revealed punched-out areas of destruction in the bodies, laminae, pedicles, and spinous processes of the vertebrae in the thoracic, lumbar, and sacral regions (Fig. 1 and 2). The intervertebral discs were intact. The vertebrae showed no evidence of collapse. The diagnosis was tuberculosis of the vertebrae.

The hospital course consisted of aspirations of the cervical mass from which thick yellowish-green pus, in which no acid-fast organisms could be found, was removed. He was given high voltage roentgen therapy to the cervical mass and to the areas of destruction in the skull. He was discharged August 27, 1941, the prognosis being poor.

The patient was readmitted to the Tuberculosis Service on December 3, 1941, because of an infiltration at the base of the right lung which appeared more definite than on the previous examinations. Aside from the diminution in the size of the cervical mass the patient had become worse. Physical examination revealed nothing new. The previous diagnosis was accepted and the patient was treated symptomatically with aspirations of the fluctuant masses. No tubercle bacilli could be isolated from the sputum or from the fluctuant masses. The patient grew gradually worse. The heart became enlarged and the possibility of a pericarditis was entertained. He died March 20, 1942.

At autopsy (performed by Drs. Lund and

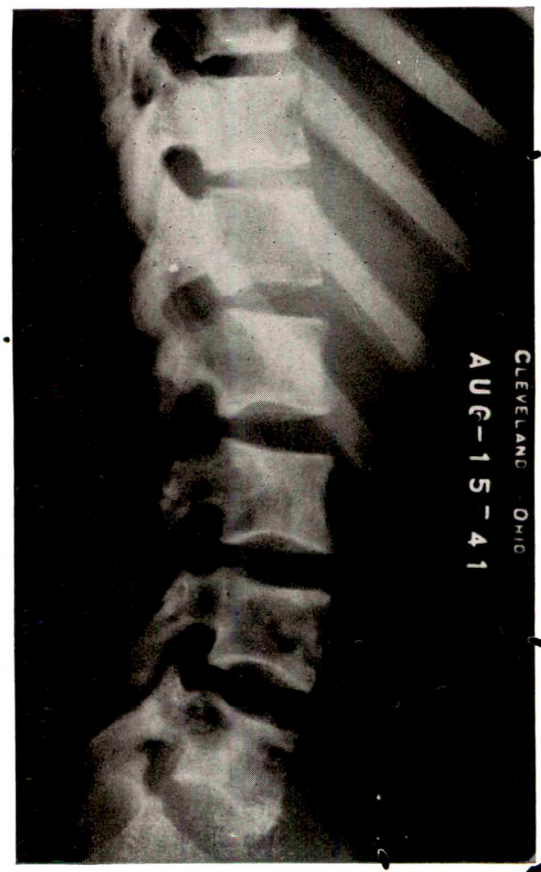


FIG. 2. Case 1. Lateral view demonstrates that areas of destruction involve the laminae and spinous processes as well as the bodies.

Kapp) a large paravertebral phlegmon which contained thick, yellow, creamy pus was present in the thoracic and lumbar regions. The anterior aspects of the bodies of the vertebrae showed deep abscess pockets involving at least one-half of the depths of the bodies in most instances. The surrounding non-cavitated vertebrae were yellow, soft and crumbly. The phlegmon extended laterally over the lateral aspects of the vertebrae. Involvement of the ribs was not demonstrated.

Microscopically, much of the bone was replaced by areas of necrotic exudate. The cytology was not distinct, but the cells appeared to be lymphocytes and polymorphonuclear leukocytes. The areas were surrounded by a fibroblastic zone. In the necrotic areas were numerous ray fungi. The organisms were numerous and had peripheral radiating clubs and central net-like mycelia.

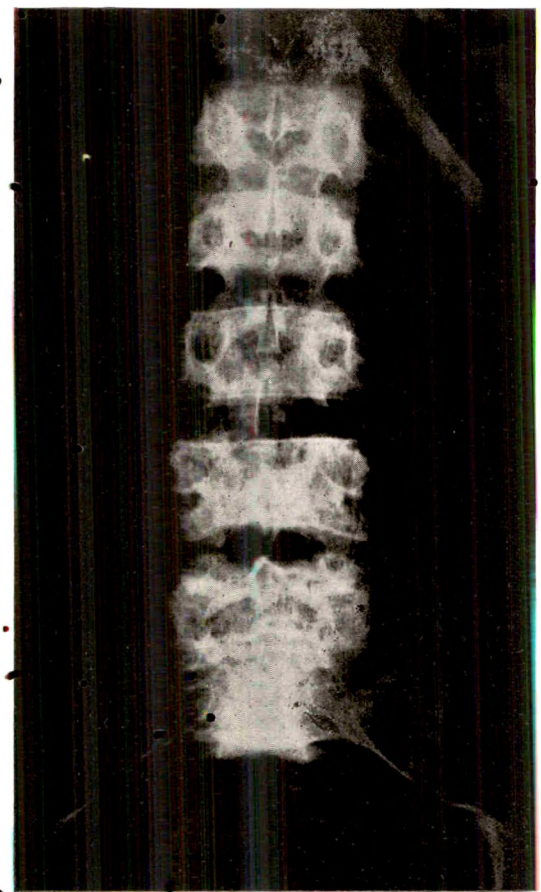


Fig. 3. Case II. Anteroposterior view shows widely distributed areas of destruction surrounded by areas of sclerosis.

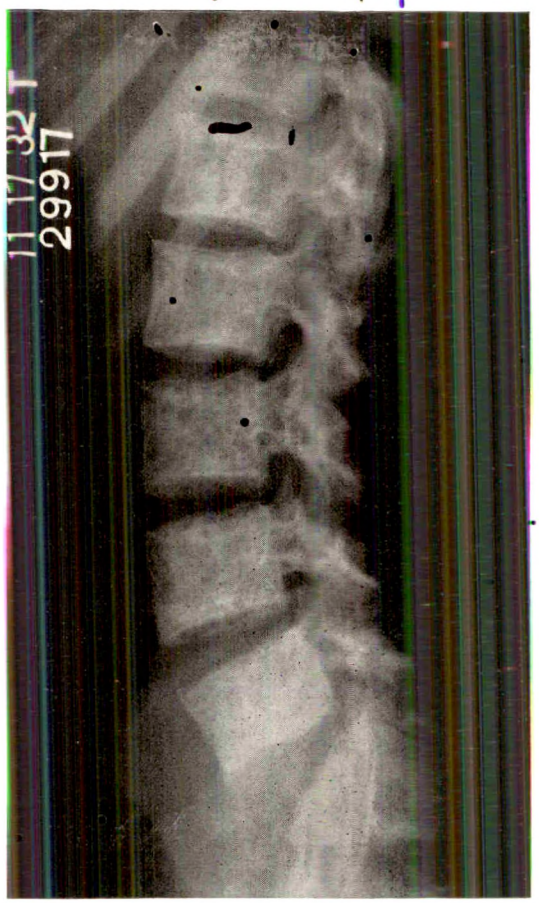


Fig. 4. Case II. Lateral view reveals involvement of all components of the lumbar vertebrae. The areas of destruction are smaller but the sclerosis is more prominent than in Case I.

The final diagnosis was actinomycosis of lungs, vertebrae, and prevertebral tissues. The lesions in the skull, ribs and tibia were not examined.

Case II. H. M., white male, aged twenty-one, entered the Surgical Service of the University Hospitals of Cleveland on December 27, 1931, with complaints of pain in the back and draining sinuses in the right lower abdominal quadrant following an appendectomy at another hospital eight months before. Nothing unusual was noted in the appendix but the diagnosis of actinomycosis had been made from the discharging sinuses.

Physical examination revealed a chronically ill patient. The teeth were in poor condition. There was spasm in both lower quadrants, especially on the right, and tenderness in the

region of the fifth lumbar vertebra. The neurological examination was negative.

The laboratory findings revealed traces of albumin in the urine and a white blood cell count of 13,000.

The diagnosis of actinomycosis was reaffirmed by another biopsy. Injections of the tracts with lipiodol and subsequent roentgenograms showed the tracts to end in the region of the cecum which was then removed surgically. Roentgenograms of the spine were negative. The sinus tracts were explored and through and through drainage instituted. The patient was discharged January 1, 1932, to be cared for at home.

The patient was readmitted November 13, 1932, with the complaint of convulsions, weakness and draining abdominal sinuses. During the interval at home he constantly grew worse. He had had two convulsive seizures and the legs had been held in a constant state of flexion for some time. There had been intermittent diarrhea.

Physical examination revealed a further increase in emaciation. The abdomen was diffusely tender and distended. There were five sinuses present in the right lower quadrant and two or three in the right lumbar region. The tendon reflexes were reduced. Sensation was normal.

The laboratory findings at this time were essentially unchanged.

Roentgen examination (Fig. 3 and 4) revealed multiple, round, radiolucent areas with surrounding sclerosis in the lumbar spine and sacrum and questionably in the ilium. The second, third, and fourth right transverse processes were markedly irregular as was the right twelfth rib. The intervertebral discs were not involved.

The patient had several convulsions while in the hospital and died eleven days after admission.

At autopsy (performed by Dr. Paul N. Harris) the abdominal organs were matted together by dense gray fibrous tissue in which abscesses could be seen. In the process of evisceration the periosteum was stripped off the vertebrae. The surface of the bone was rough and contained many small abscess cavities in which there was greenish-gray or yellow pus and destruction of the trabeculae of bone. Around the abscesses there was an increased amount of bone and the trabeculae were arranged circumferentially.

The anatomical diagnosis was actinomycosis of the left lung, liver, vertebrae, skin, subcutaneous and retroperitoneal tissues.

CASE III. J. D., white male, aged sixty-one, entered the hospital on the Medical Service on April 10, 1941, with the complaint of pain in the chest and chronic cough for two years. The pain was located in the lower portion of the left side of the chest and was most severe on coughing and deep breathing. The cough was productive only in the morning.

Physical examination revealed obvious loss of weight. The left pupil was smaller than the right. The teeth were in poor repair. Posteriorly in the left chest fine crackling râles were heard. An "e" to "a" change was heard posteriorly in the region of the fifth, sixth and seventh ribs just to the left of the spine. The liver was moderately enlarged. The spleen was not palpable. The reflexes were normal.

Laboratory examinations revealed no urinary findings of significance. The white blood cell count was 10,900.

Roentgen examination revealed a streaked infiltration extending outward from the left hilum which was interpreted as a bronchogenic carcinoma. Bronchograms revealed no abnormalities of the bronchi of the left lung.

The patient was bronchoscoped, but no mass or constriction in the left main-stem bronchus could be seen. An aspiration biopsy was noncontributory. He was treated by high voltage roentgen therapy and discharged on May 3, 1941.

The patient was readmitted on the Medical Service November 18, 1941. He continued to lose weight and his cough grew worse. His sputum had contained thick mucus, pus and occasionally blood. For the past several months he complained of a severe pain in the back.

Physical examination at this time revealed further emaciation. The lungs revealed a decrease in fremitus, râles at both bases and tubular breath sounds over the left chest. A dorsal kyphosis was present. The neurological examination was negative.

Laboratory findings were essentially unchanged.

Roentgen examination showed extension of the pulmonary infiltration on the left and an enlargement of the right hilum. The ninth thoracic vertebra (Fig. 5 and 6) was almost completely collapsed and there was questionable destruction of the adjacent ribs. The intervertebral discs were not involved. Bronchogenic carcinoma remained as the diagnosis.

The patient continued to grow worse, and developed cardiac irregularities, which were attributed to invasion of the heart by the neoplastic process. No further diagnostic procedures were done and the patient died on December 26, 1941.

At autopsy (performed by Dr. F. T. Kapp[5]) the lungs contained many soft-walled cavities filled with creamy material. Portions of the lungs were adherent to the chest wall, the mediastinum and the paravertebral tissues.

The vertebral column showed a kyphosis in the mid-thoracic region. The ninth thoracic vertebra was almost completely replaced by a cavity filled with a yellow, thick fluid. The spinal cord at the level of the ninth thoracic vertebra was soft and compressed by the collapsed bone around it. The dura in this region was surrounded by a thin, yellow layer of tissue.

Microscopic examination of lungs and lower thoracic prevertebral tissues showed the presence of a granulomatous and suppurative process containing the characteristic actinomycotic colonies. The body of the ninth thoracic vertebra was completely destroyed and the process caused a chronic inflammatory reaction in the outer layers of the dura in this region.

The final diagnosis was actinomycosis of the lungs and the ninth thoracic vertebra.



Fig. 5. Case III. Anteroposterior view shows collapse of the ninth thoracic vertebra.



Fig. 6. Case III. Lateral view shows sparing of the intervertebral discs despite the collapse of the ninth thoracic vertebra.

DISCUSSION

Three cases of actinomycotic involvement of the vertebrae have been presented. Two of the cases show the classical findings and one shows angular deformity which is only rarely described.[8] The first case probably represents involvement of the vertebrae secondary to primary mediastinal involvement. The involvement of the lungs is apparently secondary. The second case is

one of ileocecal actinomycosis with subsequent spread to the lumbar prevertebral tissues and vertebrae. The third case is one of primary pulmonary actinomycosis with spread to the mediastinum and vertebra. In the latter only one vertebra is involved in the roentgenogram.

In only 1 of the cases was there the contact with grasses and cattle. The other 2 patients did not have a history of rural occupations although the thoroughness with which this information was sought is not known.

In 2 of the cases the diagnosis was not made before death, either clinically or roentgenologically. In the case with the angular deformity of a vertebra, however, the roentgen findings were not characteristic, and even in review the diagnosis made seems to have been most proper. In the other case, the roentgen findings were sufficiently characteristic of actinomycosis to have suggested its presence. With this possibility in mind a biopsy or sputum examination might have revealed the actinomyces.

By the time the vertebrae are involved the disease is very extensive and the prognosis hopeless. All of the cases received roentgen therapy. The first case received small doses in a protracted course. The last case, however, received a full cancericidal dose.

SUMMARY

1. Actinomycosis is not necessarily restricted to persons with agricultural pursuits or other rural contacts.

2. Poor oral hygiene, a tonsillectomy, or a tooth extraction may more strongly indicate the presence of actinomycosis than a history of chewing grasses and straws.

3. When actinomycosis involves the vertebrae the roentgen findings usually are characteristic if not definitely diagnostic.

4. The roentgen signs of actinomycosis consist of areas of destruction in all portions of the vertebra, including the spinous processes and lamina, as well as the bodies, with varying amounts of surrounding sclerosis. Collapse of a vertebra despite extensive destruction is unusual, but even then the intervertebral discs are spared in the roentgenogram.

5. Three cases of actinomycosis of the vertebrae with roentgenograms illustrating the changes have been presented. Two of these cases present findings which are considered characteristic; the other shows angular deformity which is unusual.

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VOL. 51, NO. 5

HEREDITARY FACTORS IN MULTIPLE CONGENITAL DEFORMITIES

REPORT OF TWO CASES

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CASE REPORTS

CASE I. Mrs. F.G., aged twenty-one, was admitted to the A. Barton Hepburn Hospital on March 1, 1941, and on the following day, gave birth spontaneously to an infant, N.R.G., having both feet clubbed and a fluid filled sac protruding from the midline in the lumbar region. The sac, incompletely covered by skin, was assumed to be a myelomeningocele. Roentgenogram of the region (Fig. 1) demonstrates defect of fusion of the sixth, eleventh, and twelfth dorsal and all of the lumbar vertebrae. The coexistence of craniolacuna was likewise verified by roentgenogram (Fig. 2). Death of the infant occurred on the ninth day. Postmortem examination confirmed the diagnosis of myelomeningocele. The cerebral ventricles were not enlarged.

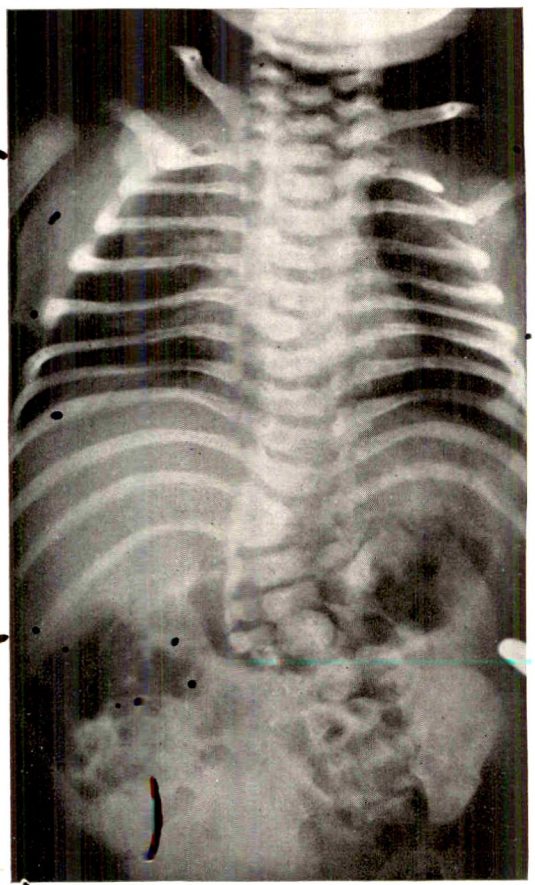


FIG. 1. N.R.G. Showing spina bifida.

The maternal patient had never had an operation, serious illness or injury. She had first

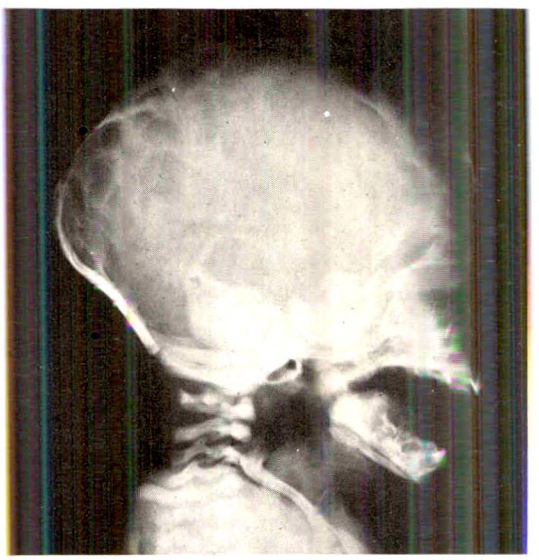


FIG. 2. N.R.G. Showing lacunar skull.

menstruated at the age of fifteen, and had been habitually regular. She was married at the age of seventeen, and the pregnancy referred to above was her third. The first was terminated by miscarriage at three months and the second went to term, the product of conception being a monster with an encephalocele. During the current admission, Wassermann and Kahn tests were found to be negative, as were those of her husband. Blood cell counts were normal, there were transiently small amounts of albumin and sugar found in urine obtained by catheterization. Physical examination on admission revealed no significant abnormality.

During August, 1941, a fourth pregnancy terminated by miscarriage at four months. The

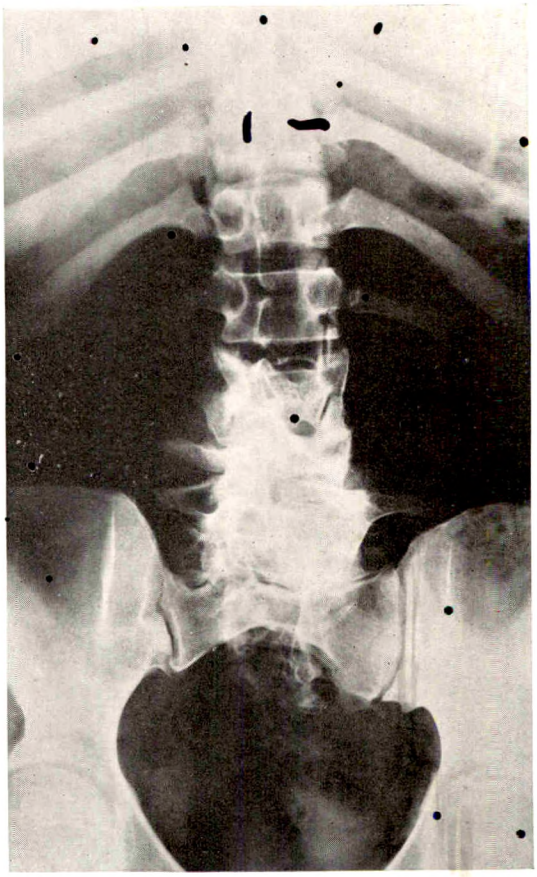


FIG. 3. Mrs. W.O., maternal aunt of N.R.G., showing spina bifida.

patient, again pregnant, came to the hospital on May 5, 1942, for roentgenographic examination, and a complete fetal skeleton was found in a position of favorable presentation. The maternal pelvis was gynecoid and the critical diameters appeared ample when measured by the Thoms grid method. On August 24, 1942, she was delivered of an infant having no physical or roentgenographic evidence of deformity.

In the family history, it was recorded that the patient's sister, Mrs. W.O., has had since birth a soft mass protruding from the lower part of her back, that she is otherwise in good health and has had two healthy children, both delivered by cesarean section. Through the courtesy of Dr. Joseph H. Wyatt of Newark, a roentgenogram was obtained (Fig. 3) demonstrating defective fusion of the second to fifth lumbar vertebrae. There was nothing else of significance in the family history. Neither parent of the sisters had ever been exposed to roentgen rays.

CASE II. Mrs. R.S., a primipara, aged nineteen, was admitted to the hospital on May 21, 1943, being then in labor. Roentgenographic examination disclosed an anencephalic fetus, which was subsequently delivered by dilating the cervix with a Voorhees bag. A postpartum roentgenogram of the fetus (Fig. 4) shows that, in addition to the deficiency of bones of the cranial vault, there is no fusion of the centers of ossification of the cervical spine. The maternal pelvis was found to be gynecoid and of ample dimensions. In the patient's past history there was only indefinite illness of childhood and pneumonia. Nothing of abnormal significance was found on physical examination or in the routine blood count, urinalysis or Wassermann reaction. In the family history, it is recorded that a second cousin (her mother's

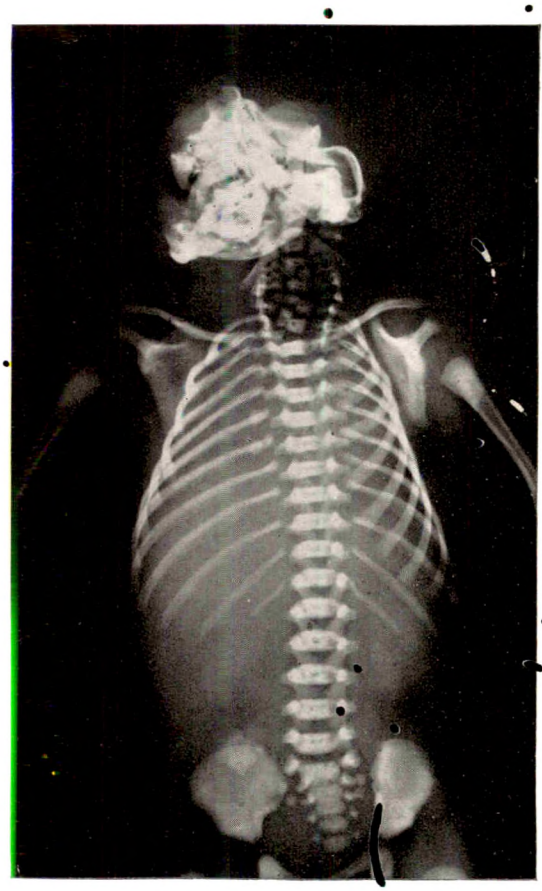


FIG. 4. Anencephalic fetus with cervical spina bifida.

first cousin had been delivered of an anencephalic fetus and that the patient's mother has six digits on each hand; her brother has six digits on one hand.

REVIEW OF LITERATURE

In presenting 2 instances of the occurrence of similar or related congenital deformities in members of the same family, the intent is to invite attention to recent medical literature offering a plausible explanation of the etiology of such deformities. Dorrance[5] attributes the defective development of the membranous bones of the calvarium in cases of lacunar skull to ischemia and increased intracranial pressure in the embryo. Bettinger[3] regards lacunar skull as a secondary effect of spina bifida, occurring when the pressure of cerebrospinal fluid over the cortex of the brain is reduced below the customary balance existing between it and the amniotic fluid, permitting the developing bones of the cranial vault to be compressed against the pulsating cerebral convolutions. The common association of craniolacuna and spina bifida is confirmed by Vogt and Wyatt,[8] who found craniolacuna to exist in 52 of 120 infants having meningocele, and 2 other cases of lacunar skull in which there was no meningocele. Shapiro and Tosti[7] concluded that dilatation of the cerebral ventricles is part of the picture of spina bifida with meningocele.

The unsupported fact of familial incidence suggests that spina bifida and the associated congenital lesions may be caused by a defect in the primary germ plasm. The comparative rarity of familial incidence would indicate that the genetic manifestation must be extremely recessive. In explanation of the mechanics involved in the formation of certain other congenital deformities Engel[6] offers a hypothesis based upon experimental observations of Bagg,[1] Bagg and Little,[2] Bonnevie,[4] and others.

Bagg and Little[2] exposed the gonads of healthy mice to moderate doses of roentgen rays and found that in the progeny of the irradiated mice there was a high ratio of animals having congenital defects of the head and extremities. These defects were carried on down through successive inbred generations though only the original progenitors received the experimental injury. The more common hereditary defects were of the eyes, varying from slight atrophy of the lids to opaque corneas and shrunken blind eyes with advanced atrophy of the optic tracts. There were somewhat less frequent defects of the extremities, including clubfoot, syndactyly and polydactyly. No such defect was found in more than two thousand control individuals of the same mouse strain. Not only was the fertility of the irradiated stock less than that of the control animals, but there were also many still-births. Blood vascular lesions were discovered about the heads of the embryos of the experimental mice.

In supplemental studies, Bagg[1] found that the embryonic lesions originally noted were at first clear fluid blebs beneath the surface, into which extravasation of blood often occurred. Blebs were found as early as the twelfth fetal day, the most frequent site being about the limb buds. Embryos were observed *in utero;* those individuals having blood blebs were marked by tail clipping, and the pregnant animal was allowed to proceed with gestation with the result that the marked animals were found to be defective while those not so marked were usually normal. Occasional visceral lesions were encountered. Bagg concluded that irradiation injured the germ plasm, that the injury produced blebs containing blood which provided a mechanism for congenital malformation.

Bonnevie[4] carried on further experimental observations with animals bred from the Bagg and Little mouse strain. She found that the clear blebs contained cerebrospinal fluid that had escaped into the subcutis through the foramen anterius, an embryonic structure in the roof of the fourth ventricle. This structure is normally a safety valve guarding against increased cerebrospinal pressure in the embryos of vertebrates. It exists prior to formation of

the foramen of Magendie. In embryos developing blebs the amount of escaping fluid or the rate of flow is increased. It was observed that the fluid had a tendency to collect in the concavities of the embryonic surface and in the ends of the limb buds; that the blebs were apt to be damaging to further development of the embryo only if there was extravasation of blood into them. A preponderance of lesions of the left side was considered to be due to the intrauterine position of right-sided twist assumed by the mouse embryo. In outcrossing experiments, Bonnevie found that modifying genes changed the specificity of the deformity.

Engel[6] lists nine congenital syndromes in which the formation of spinal fluid blebs in the subcutis of the human embryo may constitute an etiological mechanism. He finds the present differentiation artificial, and notes that all of these syndromes have in common a combination of anomaly of the skull, deformity of the extremities and a tendency to familial occurrence. The specific conditions that he mentions are: oxycephaly, acrocephalosyndactyly, dysostosis craniofacialis, Lawrence-Moon-Biedl syndrome, Morquio's disease, pleonostosis familiaris, mongoloid idiocy, craniocarpotarsal dystrophy and Turner's syndrome. These are collectively called "bleb disorders," regarded as comparable with the experimental conditions produced in the Bagg and Little mouse strain. Engel suggests that still other conditions may eventually be included.

COMMENT

While two case reports can have no statistical value, they may be used to illustrate the applicability of a hypothesis. The second case rather strikingly illustrates in successive generations congenital defects similar to those experimentally produced in mice, though no single individual in the family exhibits malformation of both the head and the extremities. The first case herein cited conforms to all of Engel's stipulations for congenital disorders of bleb origin, in that there are deformities of the skull and feet and evidence of hereditary transmission. There are also fusion defects of the spine and a congenital disorder of the central nervous system. The relatively common spina bifida is not uncommonly associated with clubbing of the feet, and it has been shown that, when complicated by meningocele, there is apt to be craniolacuna. Such diverse manifestations can hardly be attributed to intrauterine physical conditions, nor can many of them be correlated as cause and effect. While craniolacuna has been regarded as a secondary effect of meningocele, its lace-like pattern is in fact not an image of the gyri of the cerebral convexity, and it is found at an earlier age than the appearance of the usual convolutional markings.[8] All of the manifestations of clubfeet, polydactyly, spina bifida with meningocele and defective cranial development may be explained by a mechanism of cerebrospinal fluid blebs extruded into the subcutis of the early embryo. The experimental and clinical observations appear to be analogous, and it is suggested that it is entirely plausible to include these defects occurring either singly or in combination along with the conditions listed by Engel as being of bleb origin.

The animal experiments causing inheritable deformity through injury to the gonads by roentgen rays have given rise to the supposition among students of genetics that the clinical use of roentgen rays may be responsible for congenital deformities occurring in human progeny. Such disorders cannot be entirely charged to roentgen-ray injury nor is it believed to be a principal etiologic factor since the deformities were commonly seen prior to the era of clinical radiology. The primary cause of clinical as well as experimentally produced hereditary deformities can be radiant energy or another agent capable of producing sublethal changes in the primary germ cell.

The author desires to thank Drs. H. E. Clark and R. L. Stacy for assistance in obtaining family history data.

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OSTEOCHONDRITIS OF THE CAPITELLUM (PANNER'S DISEASE)

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THE more commonly encountered sites and appearance of that interesting group of osteochondropathies, termed variously osteochondritis, osteochondrosis, aseptic necrosis, epiphysitis, and chondro-epiphysitis, are well known to roentgenologists. Some of the rarer locations are understandably less familiar, and it is for this reason that this report is being made.

In 1927 Panner[1] first described the appearance of osteochondritis occurring in the capitellum, recognizing it as an entity belonging to the group that includes the more familiar sites described by Perthes, Köhler, Freiberg, Kienböck, Scheuermann, and others. In his published report[2] in 1929 he records 3 cases, 2 in boys aged ten and 1 in a boy aged seven. In a discussion of Panner's presentation, Krebs[3] reported a case of his own in a boy aged eight, and suggested the term "Panner's disease" for this condition. In 1929 Smith[4] reported a case in a four year old boy. Two cases described by Elward[5] in 1939 were both in eight year old boys. In a group of 91 endocrinopathic patients with osteochondritis in 162 areas, Schaefer, *et al.*[6] tabulate 3 instances of involvement of the capitellum. A careful search of the literature shows that these 10 cases are the only recorded instances of this condition. Doubtless it is not so rare as this would lead one to suspect, since its presence is probably overlooked in numerous instances. The following case was recently seen by the author:



FIG. 1

D. S., male, aged eight, was perfectly well until three weeks prior to the examination. At that time, in the act of throwing a dart at the dart-board, he found he could not completely flex the right elbow. There was no previous history of any trauma. Since then he has developed a limitation of complete extension and complete flexion of the elbow (due to moderate



FIG. 2

forded by a sling, the clinical symptoms and signs disappear over a period of a few weeks. The roentgen manifestations are not commensurately improved, however, and may persist for one to three years before complete restitution is seen. This benign course resulting in practically complete restoration of structure and function follows the familiar pattern in osteochondritis where weight bearing is not a factor. Theoretically, however, such an elbow may possibly be more susceptible in later years to the development of secondary hypertrophic or traumatic arthritis. No case has been followed long enough to determine this.

SUMMARY

Attention is called to an apparently rare site of osteochondritis, the capitellum, and another case is added to the very few reported in the literature.

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discomfort) although the intermediate range of motion is free and painless, and pronation and supination are not interfered with. A slight swelling about the joint, particularly over the lateral aspect, has developed, but at no time has there been any redness. Figures 1 and 2 are views of the involved elbow, while Figures 3 and 4 show the uninvolved elbow for comparison.

From the data available in the recorded cases, osteochondritis of the capitellum may be summarized as follows: Clinically, the patient is a male (all of the cases in which sex was recorded were males) seven to ten years of age. There may be a history of a relatively mild preceding trauma or there may be merely a gradual onset of discomfort in the elbow. In the latter case the trauma may have been so insignificant that it is not recalled. The etiology of osteochondritis in general is still a debatable matter and is outside the scope of this paper. In any event, the discomfort is only moderate and accompanies chiefly attempts at extreme flexion or extension, which are limited. There is a slight swelling of the joint, but no redness.

The changes that are seen roentgenographically consist of an irregularity of contour of the capitellar epiphysis. The cortex is thin and there is a subcortical zone of rarefaction, as can be seen in Figures 1 and 2. There may be interruptions in the cortical continuity. Within the epiphysis

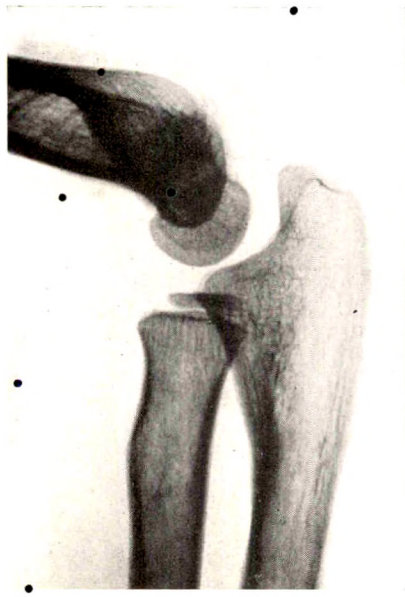


Fig. 4

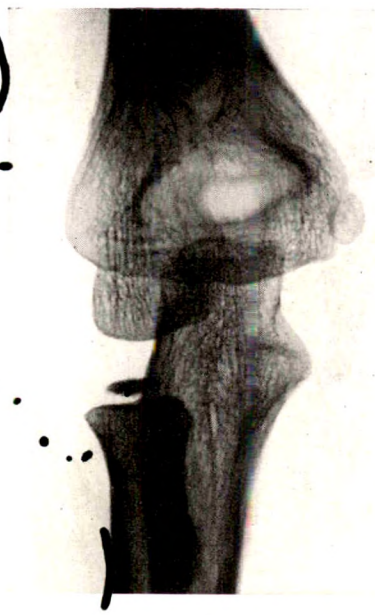


Fig. 3

are seen irregular zones of bony condensation. In marked cases the epiphysis undergoes fragmentation. These changes are the ones generally seen in osteochondritis in any site. The epiphysis, as a result of these alterations, may appear smaller than normal and the joint space between the epiphysis and the head of the radius appears greater than normal. Some soft tissue swelling may be seen about the joint, especially on the lateral aspect. This condition once again demonstrates the advisability of making bilateral examinations of the joints in children, since the changes are readily apparent when compared to the normal, but if the condition is an early one, it can easily be overlooked by someone not completely familiar with the normal appearance of the epiphysis.

With rest and relative immobilization af-

VOL. 51, NO. 6

ROENTGENOLOGICAL FINDINGS IN BILATERAL SYMMETRICAL THINNESS OF THE PARIETAL BONES (SENILE ATROPHY)

REPORT OF A CASE WITH A REVIEW OF THE LITERATURE

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BILATERAL symmetrical thinness of parietal bones, also known as senile atrophy of the parietal bones, biparietal thinning, symmetrical thinness of the parietal bones, and so forth, is well known in the anatomical, anthropological and pathological literature but has received little attention in that devoted to roentgenology.

HISTORICAL

In all probability the condition was known to the ancients, since many examples taken from Egyptian tombs have been described.[14,48,56,57] Chiari[9] ascribes the first modern description to Köhler[29] in 1786. Descriptions began to make their appearance in the European literature with Voigtel[63] in 1804 and thereafter increasing numbers of reports appeared. Ribes[42] in France and Humphry[23] in England were among the early writers. In the Western Hemisphere, Shepherd,[54] first as Demonstrator and later as Professor of Anatomy[55] at McGill University recorded the first cases found in North America. Willard[66] described the first cases found in the United States in 1905. It was not until 1926 that Moore[34] in this country and Casati[7] in Europe published papers with illustrations of roentgenological findings.

SITE OF INVOLVEMENT

The characteristic location of cranial thinning is in the parietal bones between the sagittal suture and the parietal prominence. Earlier authors, notably Humphry,[23] claimed it could occur only in that region. However, subsequent experience has proved this contention erroneous; in Bloch's[4] case, both temporal bones were involved and Chiari's[9] had implication of the parietal, temporal, frontal and occipital bones on both sides. Greig[19] illustrates one example of thinning of the planum occipitale and Casati[7] shows a solitary depression which crosses the sagittal suture somewhat anterior to the bregma. The latter is the only example we have been able to find in which the sagittal suture was actually crossed; in no case of bilateral thinning has this occurred.

Characteristically there is a crest-like ridge of intervening bone, centered on the sagittal suture which separates the parietal depressions; frequently there is a shallow groove-like depression of the sagittal crest —this feature is illustrated in the postero-anterior roentgenogram of our case.

TYPES

Greig[19] described two chief forms: (1) a roughly triangular or quadrilateral flattened depressed area, and (2) a trough-like depression running longitudinally along both sides of the sagittal suture; in the latter type, extension onto the frontal and occipital bone is likely to be found.

PATHOLOGICAL CONSIDERATIONS

There is no sharp line of demarcation between the depression and adjacent normal external tables; the margins shelve gradually into the thinned area. The walls of the depression are usually dark due to blood-containing medullary spaces,[17] and may be porous, though usually there is sclerosis. The floor of the depression is usually white, in sharp contrast to the darker color of the walls, and frequently it is of parchment-like thinness, extremely fragile and translucent. An excellent example of this feature is shown by Pancoast, Pendergrass and

Schaeffer[38] in a specimen from the Warren Museum of the Harvard Medical School.

Humphry,[23] Durward,[13] Greig,[19] and Hill[21] have drawn attention to the fact that the parietal foramina are spared; upon approaching them, the thinning process skirts around and leaves a margin of bone about a centimeter in width.

Penetration of the thin floor may take place with consequent exposure of the dura. Virchow[61] was probably the first to point out that the process commences in the superficial layers of the external table and gradually progresses through it, the diploe and, on occasion, the inner table. Previously Rokitansky[45] and Lobstein[30] thought that the diploe disappeared first, thus allowing the inner and outer tables to come together and coalesce. Sauvage[48] thought that the process started with the disappearance of the meningeal lamina of the inner table; thereafter it caused absorption of the layers of the external table next to the diploe whence it spread through the more superficial layers of the lamina externa and then to the diploe. There appears to be little support for his ingenious theory. Virchow's explanation gains support from the fact that the defects are always widest at their external lamina aspect.

Virchow[61] was the first to study ground sections microscopically, and his findings have been confirmed in more recent years by Ziegler[67] and Schmidt.[50] According to them the process starts by localized disappearance of the superficial layers of the external lamina. Immediately beneath the absorbed region the bone tissues become more stratified than usual and become sclerotic by apposition of lamellar bone spicules in the diploic spaces. Subsequently this stratified sclerotic zone disappears by absorption, and that which was normal, lying immediately beneath, becomes sclerosed. In this manner the process extends progressively through all the cranial layers.

Schmidt[50] was impressed by the lack of osteoclasts and came to the conclusion that the process is one of diminished bone formation rather than of increased bone destruction.

ETIOLOGY

Various theories have been advanced in attempts to explain this curious thinning. Ferré[16] states that Oribase and Fallopius attributed the thinning to baldness, the deficiency of hair permitting emaciation of soft parts and of bone. By the majority of authors it is considered an atrophic change, but explanations as to the cause of the atrophy vary widely.

Virchow[61] is generally credited with being the first to suggest the theory of senile atrophy. As a matter of historical fact that distinction belongs to Lobstein,[30] and Virchow recognized his priority; Rokitansky[45] and Maier[31] both adopted this theory before Virchow. Many other authors[7,16,25,28,37,50,54] have felt it the logical explanation. In the main, evidence of skull senility has been felt to depend upon (*a*) marked degree of decalcification, (*b*) generalized diminution in width of the tables, (*c*) suture obliteration, (*d*) edentulous condition of the maxilla with atrophy and (*e*) osteoporosis of the skull tables. Ashley-Montagu,[1] in a full discussion of the question of skull aging, concluded that suture closure and state of dentition are the more utilizable guides for determining age. Topinard[59] found parietal thinness and non-closure of sutures in the skull of a man between sixty and seventy years of age; Greig[19] found thinness of both parietals in a skull which still showed traces of the metopic suture and had full dentition in a good state of preservation. In our case all sutures, including the metopic, are visible and the upper teeth are well preserved. It would thus appear that the "senile" theory cannot be accepted as the cause in all cases.

As a variant of the senile theory, Carriere[6] and Weber[63] considered it due to excessive physiological atrophy. The latter concluded that the involved area has no muscle attachments and is mechanically of little value "for maintaining the strength of the brain case."

Syphilis, as might be expected, has been held responsible by many authors. Paget[36] was among the first to form this opinion but he later became converted to the senile atrophy theory. Rokitansky,[45] Topinard,[59] Hutchinson[27] and Meyer[32] later adopted this theory, though Meyer was not enthusiastic. Hutchinson appears to have had some justification because his patient was known to have had a chancre. Modern writers,[41,43] reporting serological findings in their cases, have found no evidence of lues, and in our case the Wassermann and Kline reactions were negative.

Sauvage[48] based his views upon the study of 41 cases which formed the basis for his thesis and concluded that interference with vascular supply was the probable cause. In 1870 Carriere[5] expressed the opinion that the change is due to atheromatous vascular changes. Broca[9] lent some support to this theory when he stated that the posterior parietal region is the last to ossify and that vascular nutrition is least in this part of the skull. Shepherd,[55] in attempting to explain the vascular theory, held that the temporal arteries which supply the external table, being uncovered by muscle, are more apt to become involved by senile change and so interfere with nutrition. This type of change is less likely to affect the middle meningeals which supply the inner table. Durward[13] and Hill[21] were inclined to support this theory. In comment it should be observed that there have been no reports of careful dissection and microscopic studies of the vascular supply to the parietal bones in cases of thinning. Until such studies have been made, the theory is open to question.

Humphry[23] proposed the theory of congenital defect but later[25] acknowledged he had insufficient evidence to support it, and favored that of senility. Shepherd[55] had a case, unproved, in a woman who stated that her depressions had been present since she could remember and that her father had had similar depressions. He concluded that the condition could be both congenital and hereditary. Bloch,[4] and Roger and Schachter[47] have been among those who accepted this theory.

Chiari[10] stated that Després[12] originated the theory that "planing" action of the galea aponeurotica across the parietal eminences might cause a gradual wearing away of the external table. Humphry,[26] abandoning his two previously held theories, evidently believed this viewpoint original with himself. Chiari[10] concluded that this was the most probable explanation. Greig,[19] on the contrary, considered it beyond the power of the aponeurosis to produce such a change and G. E. Smith[56] was of the opinion that the parietal bones would be more likely to modify the endon. The fact that the defects occur in parts other than the parietal bones is further evidence against this theory.

Meyer,[33] Rossbach,[46] and Hollander[22] were the chief exponents of the theory that mental disorders, notably melancholia, can cause the deficiencies. While unbiased writers have reported mental instability[58] and headaches,[34,41] and though our patient is subject to attacks of vertigo, the number of patients showing reliably reported mental signs is too few to consider this the sole cause. Moreover it is not unlikely that the symptoms in many cases are a manifestation of the individual's period of life.

Smith[56] found 70 cases among Egyptian mummies of the wealthy class who lived between the Fourth and Nineteenth Dynasties. He noted that people of that class and in that period were in the habit of wearing heavy, full-bottom wigs and concluded that continuous pressure from the weight of such an object was the cause. While this may have been the cause in the particular class of people to whom he had reference, it cannot be considered so among the cases from Europe and this country reported since the middle of the nineteenth century. A further objection lies in the fact that the thinning does not always occur at the parietal eminences.

Greig[19] has been followed by Cave[8] and

Pancoast, Pendergrass and Schaeffer[38] in support of the so-called "dysplasia" theory. According to this view "in the centre of the affected parietal area the diploe has never developed, and this part of the parietal has remained in the same condition as the lower parts of the inferior occipital fossae where the bone is thin, semi-transparent and destitute of diploe" (Greig). Later in life, as the surrounding diploe develop, the area of abiotrophy[19] remains as a depression. Greig's arguments are convincing and his theory could explain the known cases.

INCIDENCE

The abnormality is not confined to the human race. While Pick's[40] cases among dogs were of the generalized type affecting all of the skull bones, Humphry's[24] example in an adult female orang-outang was typical of the groove or trough defect found in man.

Racially, a large number of cases have been reported among ancient Egyptians;[14,48,56] this preponderance is probably more apparent than real since large quantities of concentrated material have been made available through archaeological investigations. Many cases have been reported among Europeans of England, France, Italy and Germany.[25,19,27,48,15,16,7,41,34,10,61] In the United States, Moore,[34] Meyer,[32] and Willard,[66] and in Canada, Shepherd[54,55] have reported cases. Durward[13] had 3 cases among the aborigines of New Zealand and 2 in Chinese. His 2 cases, with ours and that reported by Davis and Thurnam[11] comprise the 4 cases found among this race. Hill[21] had a case in a Singhalese. In all probability the condition is more common than is generally realized and doubtless more cases will be reported from time to time. There appear to be no racial limits.

Regarding incidence according to sex, it is difficult to draw accurate conclusions. More than two hundred cases are included in the literature reviewed herein, and in only 69 was the sex recorded; of these, 45 occurred in females. It is quite possible that accurate records might either modify or reverse the proportions.

According to age, Shepherd's[55] case in a forty year old male appears to be the youngest of those whose age was definitely known. Smith[56] found examples in skulls which he considered to be not under twenty-five or possibly thirty years of age. The upper limit seems to be restricted only by the natural span of life; Sauvage[48] had one example in an individual who had attained eighty-eight years.

FREQUENCY

Durward[13] found 5 cases in one thousand dissected skulls. Sauvage, in an investigation of the museums of Paris, found 28 examples in two thousand skulls. Smith and Jones[57] found 7 cases in ten thousand skulls. During the past five years we have seen skull roentgenograms of more than five hundred different patients of various age groups and this is the first example to come to our attention. While statistically our material cannot be considered sufficient to be significant, the conclusion appears to be warranted that the condition is not common among the average population of this country.

IMPORTANCE

While, in view of its apparent rarity, the abnormality under consideration may be considered as more of a curiosity than of clinical importance, two writers[26,66] have reported cases of death following fracture through such thinned areas. A comparatively trivial head injury might conceivably result in extensive brain laceration from penetration of fine splinters of bone. A head injury which would cause no more than a bruise in an ordinary individual might result in death to one whose skull had these thin spots. The legal complications could be extremely involved and interesting.

ROENTGENOLOGICAL CONSIDERATIONS

There is a singular dearth of references and illustrations in the roentgenological literature. Schüller[51,52] and Grashey[18] state

that interesting roentgen appearances may be found but do not include illustrations. Hempel[20] does not mention it at all. Roger and Schachter[44] associate the thinning with parietal lacunas in a descriptive paper but do not show illustrative roentgenograms. Pancoast, Pendergrass and Schaeffer[38] show photographs of specimens but do not include roentgenograms.

Moore[34] and Casati[7] illustrated their reports with reproductions of roentgenograms but showed only frontal views in which a thin bridge of bone covered over the defects. Judged upon the basis of their illustrations the cases could, with justice, be considered examples of enlarged parietal foramina but for the fact that the individual patients were adults, and, in such, enlarged parietal foramina are usually sharply defined, circular radiolucent areas.[38 (Fig. 5)]

Raviola[41] and Rizzo[43] illustrate their case reports with reproductions of roentgenograms identical in appearance with ours in both posteroanterior and lateral projections.

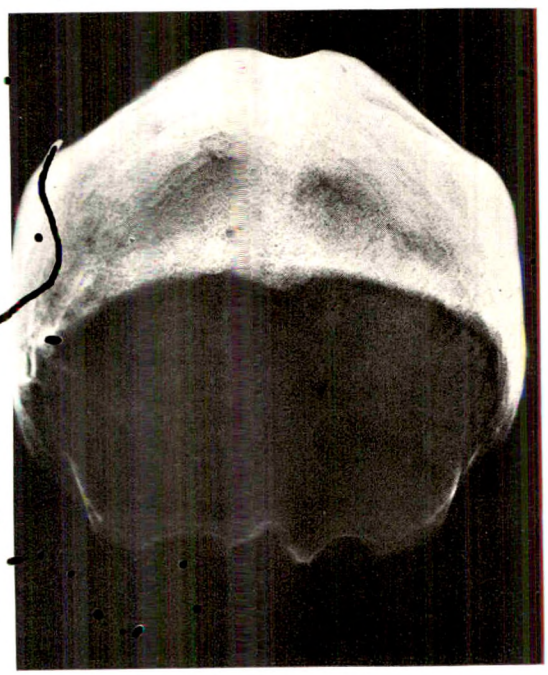


Fig. 1. Tangential view of specimen No. 12640 in Warren Museum, Harvard Medical School. Compare with Figures 3 and 4. (Figures 1 and 2 are reproductions from roentgenograms furnished the author by Drs. M. C. Sosman and M. S. Donovan.)

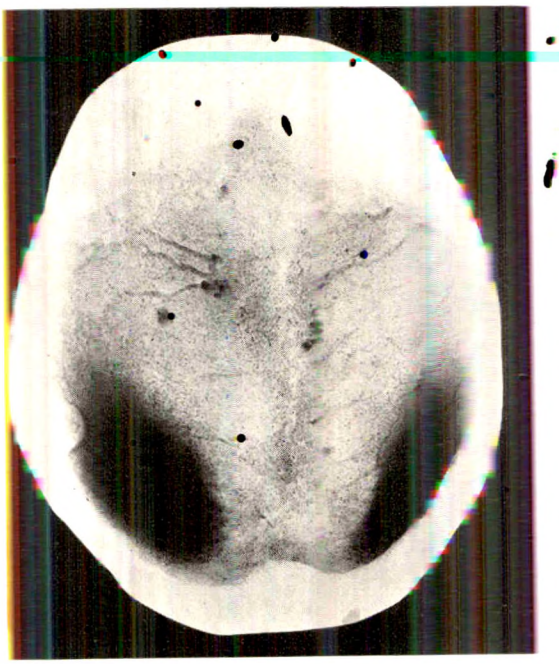


Fig. 2. Superior-inferior view of specimen No. 12640 in Warren Museum, Harvard Medical School. (The photographs of this specimen are reproduced in Pancoast, Pendergrass and Schaeffer,[38] page 11, Figures 13 and 14.)

Since the illustrations of Moore, Casati, Raviola and Rizzo were from living persons upon whom no dissection studies had been done, we felt it advisable to prove beyond reasonable doubt that our case is indeed bilateral symmetrical thinness of the parietal bones. Since our patient is now in excellent health and prospects of autopsy proof consequently remote, it became apparent that the diagnosis would require support from analogy to known anatomical material upon which roentgenographic studies had been carried out. Through the great kindness and cooperation of Dr. Merrill C. Sosman and his associate, Dr. M. S. Donovan, I was furnished with tangential and superior inferior roentgenograms of the specimen (No. 12640) in the Warren Museum of Harvard Medical School; the photographic illustrations of this skull cap are published in Figures 13

and 14, page 11, of "The Head and Neck in Roentgen Diagnosis."[38] Photographs of these roentgenograms are reproduced herewith as Figures 1 and 2. By comparison with our Figures 3 and 4, the similarity will become apparent.

DIFFERENTIAL DIAGNOSIS

In a given roentgenographic study it will become necessary to differentiate symmetrical bilateral thinness of the parietal bones from important pathological conditions. Neoplastic disease may be excluded from the finding of (1) a symmetrical bilateral localized involvement with (2) smooth, regular margins, (3) absence of a surrounding zone of new bone formation, (4) lack of evidence of malignant lesions elsewhere in the body and (5) lack of pain or tenderness. Pressure erosion of an intracranial tumor would cause the defect to be wider at the inner than at the outer table of the skull, and it would be unusual to find the defects symmetrical as is the case in bilateral thinning; furthermore, in disease so advanced one would expect pronounced intracranial roentgenological manifestations with symptoms referable to the brain. In bilateral symmetrical thinness there is no increase in skull size, increased thickness of skull tables or "woolly" appearance characteristic of osteitis deformans. In addition, the skeleton elsewhere will be negative for evidence of this disease. Syphilitic involvement may be excluded by absence of positive serological findings, absence of porous or "moth-eaten" bone appearance, sequestration and periostitis. It is to be differentiated from osteomyelitis by lack of history of preceding inflammatory disease of the scalp, sinuses, mastoid or osseous system elsewhere and from lack of history of skull trauma. There is no periostitis, sequestration or surrounding bone reaction in bilateral symmetrical thinness, and fever, pain and tenderness are absent. From congenital defect such as osteogenesis imperfecta it may be differentiated by the appearance of bone which presents the impression of having become modified locally after having been normally formed, by no increase in width of the sutures, absence of enlargement of the head and lack of abnormal skeletal changes. Examination of the scalp with determination of absence of operative scars will suffice to exclude the possibility of trephine openings.

Enlarged parietal foramina differ in that they are openings through both tables of the skull and more radiolucent than thinned areas. The parietal foramina are usually situated nearer the midline than are areas of symmetrical thinning and on palpation, pulsations may be felt. Enlarged parietal foramina show a hereditary tendency,[38] and this may be the most important differential feature when one has to deal with diagnosis of a case of bilateral thinness in which both tables have become perforated.

CASE REPORT

C. L. W., a male Chinese, aged sixty-nine, was admitted to St. Vincent's Hospital on November 7, 1941, as a patient of Dr. George Schenck, after having been struck by an automobile. He sustained fractures of the sixth and eighth right ribs, an injury to his right hand, lacerations of the scalp, and shock. The presence of rib fractures was confirmed by roentgenological examination; there was no fracture of the hand.

He was born in California but as a young man had lived for many years in China where, according to custom, he had worn a pigtail. However, for the past thirty years he had lived in Norfolk. He had been in excellent health all his life and had never before been a patient in a hospital. About three months prior to admission he had had his lower teeth extracted because of caries. For about three years he had had attacks of vertigo which came on while walking. Inquiry into bodily functions failed to elicit any indication of abnormality; specifically he denied any dysuria, digestive, pulmonary, cardiovascular or neurological symptoms.

He has three sons, one* of whom lives in

* This son is forty-five years of age and was born in China. He has always been in good health and owns his own laundry business. To palpation he presented posterior parietal "soft spots" similar in all respects to those of his father except that they were smaller. For superstitious reasons he has persistently refused to permit roentgenological examination of his skull.

Portsmouth, Virginia, the others live in California.

Blood pressure on physical examination was 114/72. Temperature, 96° F.; pulse 74, regular in rate, force and rhythm; respirations 26.

Apart from tenderness associated with the rib fractures and consequent limitation of respiration on the right side, the thorax, lungs and heart showed no abnormality. The abdomen was soft, without localized tenderness or masses. The prostate was small and fibrosed. The penis was normal as were also the testicles. The eye grounds were normal and there was no abnormality of neuromuscular reflexes.

The interesting physical findings were in connection with the skull. On either side of the midline, in the posterior parietal region there were palpable two "soft spots" each measuring approximately 4.5 by 6.5 cm. in greatest diameter, having their long axis parallel to the sagittal line. To palpation they gave the impression of being depressions whose margins shelved rather abruptly downward; there was no surrounding elevation of bone. In their central portions one palpated a pad of soft tissue but from the sense of resistance one received the impression that they had a bony floor. There was no localized soft tissue swelling over these areas and the patient made no complaint of tenderness on palpation. No pulsations or fluctuation could be detected and, when steady pressure was applied over them, there was no alteration in blood pressure. These "soft spots" were situated between the midline and parietal prominence on either side, were symmetrical in location and were separated from each other by a bony ridge approximately 5 cm. in width which centered on the sagittal suture. There was no evidence of scar from a surgical operation anywhere in the scalp. The hair showed a tendency to thinness over the frontal and temporal regions but was moderately thick over the parietals and occipital.

In response to direct questioning the patient stated that he had known about these "soft spots" for two or three years and he associated them with his attacks of vertigo. It was not possible to prove any connection and no cause was found for his stated unsteadiness.

The skull and scalp were otherwise negative for any abnormal findings on clinical examination. The maxillary teeth were present and, apart from crowding of the central incisors,

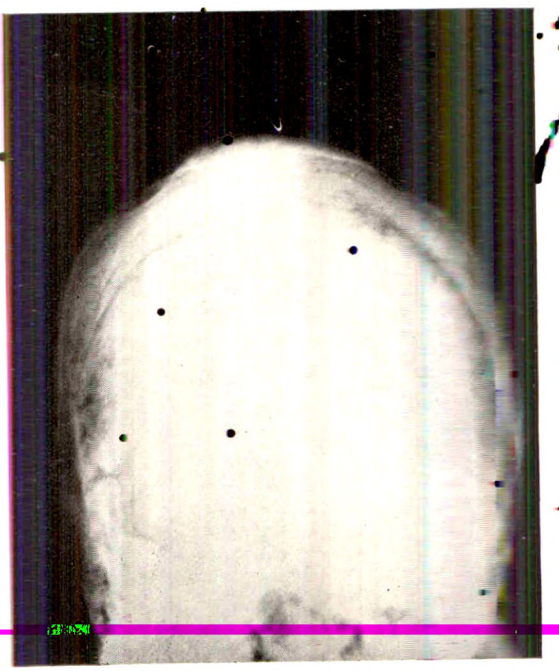


Fig. 4. Basal (parieto-occipital) roentgenogram of the case reported here. Note similarity to Figures 1 and 2.

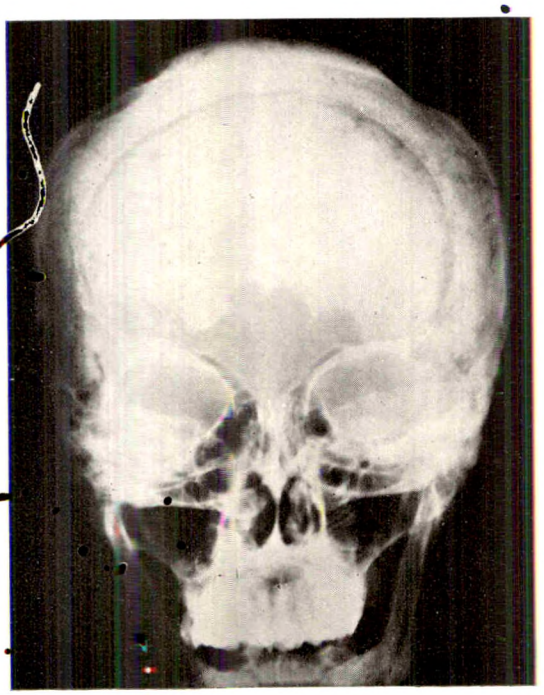


Fig. 3. Posteroanterior (nose-forehead) roentgenogram of the case reported here. See text for detailed description.

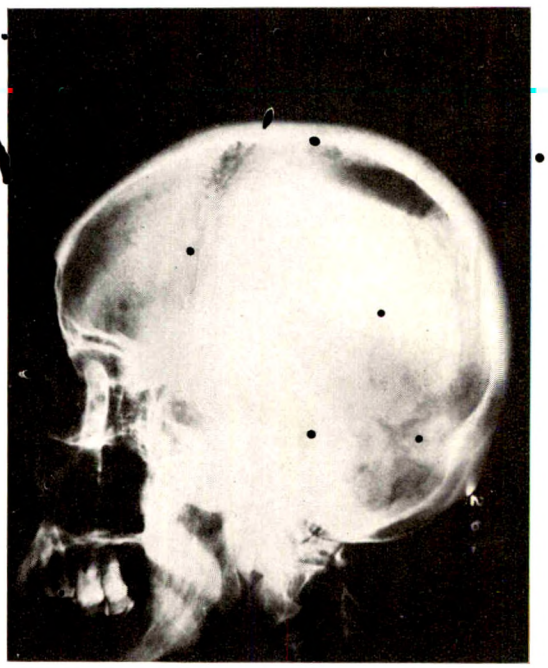


FIG. 5. Lateral roentgenograms of the case reported here.

were apparently not abnormal; the lower jaw was edentulous, and showed no evidence of inflammation or other abnormality.

Because of persistent vomiting, frontal scalp swelling associated with lacerations and vague head pain—all attributed to the accident—the patient was referred to me for roentgenological examination of the skull. The following is a description of the findings which are illustrated in Figures 3, 4 and 5.

In posteroanterior (nose-forehead) and basal (parieto-occipital) projections there were bilateral excavations of the external table and diploe, symmetrically placed on either side of the midline and separated by apparently normal bone approximately 6 cm. in width. These depressions were about midway between the sagittal suture and parietal eminences. The depression on the left side was slightly broader than that on the right—5.0 cm. and 4.5 cm. respectively. The depth, from pericranial line to floor, was 2.0 and 1.8 cm. for the left and right depressions, respectively. The floor of each depression was represented by crossing convex-concave linear bone shadows, considered to represent the superimposed meningeal surfaces of the inner table and the floor of the depression; no evidence of perforation could be detected. There was a shallow, midline grooving of the sagittal suture and adjacent bone on either side, measuring approximately 1 cm. in width by about 3 mm. in depth.

In lateral projection (Fig. 5) the defects were visible as ovoid zones of increased radiolucence in the posterior parietal region on either side; each was about 6.5 cm. in sagittal length. The margins were not sharply defined but appeared to shade gradually into the density of surrounding skull tables. The defects were smooth in outline and presented no localized irregularities; there was no evidence of periosteal reaction or of sequestration.

Just anterior and medial to these defects there were small, irregular areas of increased radiolucence which were judged to represent depressions for pacchionian bodies; there was no evidence of connection between these and the larger defects described above.

In the frontal region the calvarial thickness was approximately 0.8 cm.; over the depressed areas (in lateral projection) it was approximately 1.0 cm., and from that point to the inion there was a gradual increase in thickness. At no point was there any suggestion of abnormal skull thickness. There was no irregularity of calcification, and nothing indicative of osteitis deformans, neoplastic or inflammatory disease could be seen. A few localized areas of rarefaction, judged to be indicative of slight osteoporosis, were seen.

There was no evidence suggestive of fracture, either recent or old.

By strong transmitted light the scalp outline could be seen in the original roentgenograms, and there was no evidence of local increase in thickness; over the parietal regions lateral to the depressions it was approximately 0.6 cm. and over the sagittal crest it was approximately 0.9 cm. thick. Soft tissue filled in the depressions but there was no bulging above the external scalp line.

There was no evidence of increased intracranial tension. All of the sutures, including the metopic, were visible and not widened. The diploic vessels were not unduly prominent nor were their grooves unusually deep. The sella turcica measured 0.8 cm. in depth and 1.2 cm. in length and showed no evidence of erosion. The pineal body was not sufficiently calcified to be visible.

The paranasal sinuses showed no indication of abnormality other than moderate thickening of the maxillary antral mucosa. The maxillary

teeth were present; the mandible was edentulous.

Roentgen examination of the entire skeleton failed to show any evidence of inflammatory, neoplastic or other pathological involvement. Particular attention was paid to the spine, pelvis, femurs and tibiae but no evidence of osteitis deformans could be detected.

There were bilateral renal and right ureteral calculi; the solitary left renal calculus was in the lower superior minor calyx and measured 0.4 by 0.6 cm. There were two right renal calculi, both in inferior minor calices; the smaller (lower) one was 0.3 cm. in diameter and the larger, about 1 cm. above it was 0.4 by 0.9 cm. in size. The left renal drainage system was sharply defined and was judged to be normal. There was slight dilatation of the superior calices and renal pelvis on the right side. The right ureteral calculus measured approximately 0.9 by 1.5 cm.; it was ovoid in outline and had caused dilatation of the ureter down to a point opposite the middle of the sacral wing; it was movable within the ureter. Dr. Devine noted no obstruction on catheterization of this ureter, however. The renal shadows were normal in size and the psoas shadows were sharply defined.

The patient persistently refused to admit tenderness of either renal or ureteral region and denied any symptoms referable to the urinary tract.

Culture of urine from the left kidney was sterile after forty-eight hours; the right specimen showed *Staphylococcus haemolyticus* after forty-eight hours.

Roentgen examination of the lungs and heart revealed no abnormality; there was no suggestion of pulmonary tuberculosis or neoplasm.

The gastrointestinal tract was investigated roentgenologically by ingested barium sulfate suspension and by barium enema. There was no evidence of malignancy or other abnormality.

Clinical Course. The patient's recovery was somewhat slow, chiefly owing to the fact that he could not make himself understood and could not properly understand what was said to him without the aid of an interpreter. On November 22 he had a sharp rise of temperature to 105° F., pulse to 120 and respirations to 36. It was thought that he had some respira-

Laboratory Findings.

Blood	Nov. 8	Nov. 12	Nov. 23
Hemoglobin	90%	90%	91%
Erythrocytes	5,500,000	4,600,000	4,880,000
Leukocytes	10,100	13,900	37,850
Polymorphonuclears	71%	71%	71%
Large lymphocytes	22	22	[illegible]
Stab	3	4	12
Transitionals	3	2	Myelocytes [illegible]
			Metamyelocytes [illegible]
			Marked toxic granulations

Sedimentation rate	1st hour	20
	2nd hour	60
Blood calcium	Nov. 12	10.0 mg.
	Nov. 16	11.0 mg.
Wassermann and Kline	Negative	

Urinalysis—all specimens voided.

	Nov. 8	Nov. 12	Nov. 18	Nov. 24
Color	Yellow	Amber	Amber	Cloudy, red color
Reaction	Neutral	Acid	Acid	Alkaline
Specific gravity	1.008	1.015	1.015	1.017
Albumin	Negative	Negative	Trace	Trace
Sugar	Negative	Negative	Negative	Negative
Mic. pus			Few	
Mic. blood			Occasional	Few
Epith.	Occasional			
Crystals				Triple phosphate and amorphous phosphate

tory distress and a diagnosis of pneumonia was considered but could not be proved by clinical or roentgen examinations. The cause apparently was dietary since, when fluids were forced and he was placed on a high caloric diet, immediate improvement resulted and his temperature, pulse and respirations returned

ness of the parietal bones has been presented.

2 Evidence in support of this diagnosis has been submitted; briefly this evidence consists of:

(a) Analogy between roentgenological

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VOL. 51, NO. 6

THE ENCEPHALOGRAPHIC APPEARANCE IN TWO CASES OF PONTINE GLIOMA IN CHILDREN

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IN GLIOMA of the pons the early signs are cranial nerve palsies, and increased intracranial pressure is a late development. For this reason, the neurologist, at times, has difficulty in distinguishing between cases of glioma of the pons and encephalomyelitis. At the Cleveland Clinic encephalography has been performed in 2 cases of glioma of the pons, and the appearance was found to be characteristic.

The 2 cases in which we are reporting the pneumo-encephalographic appearance were children three and eight years of age. In both of the cases the diagnosis was either encephalomyelitis or a pontine neoplasm. There was no evidence of increased intracranial pressure in either case, and accordingly air encephalograms were made.

The three year old girl (Case I) died in this institution a short time later, and a postmortem examination confirmed the encephalographic diagnosis. There was a marked nodular tumefaction of the pons, more pronounced on the right side. Sections through the pons and cerebellum showed marked swelling of the pons, causing a posterior dislocation of the floor of the fourth ventricle. Microscopic sections showed the tumor to be a spongioblastoma polare. This specimen is shown in Figure 4*A*.

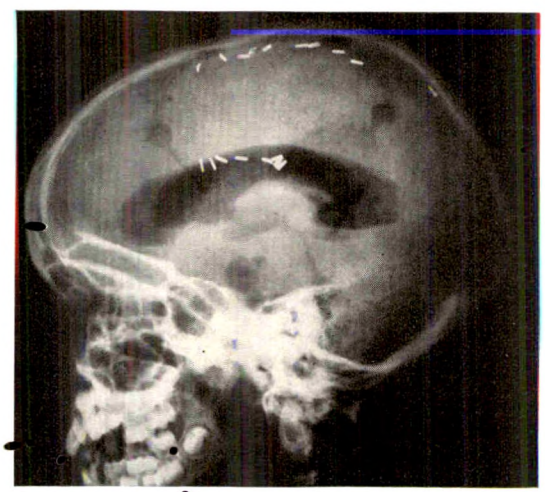


FIG. 1. This encephalogram was selected because it depicts well the normal contour of the pons. It is the case of a child who had been operated upon for a subdural hematoma. The pons is well outlined, bounded posteriorly by the fourth ventricle and anteriorly by the pontine cisterns. The thickness of the pons is 2.3 cm. The floor of the fourth ventricle is flat or perhaps slightly concave, and the normal curve of the aqueduct is well shown.

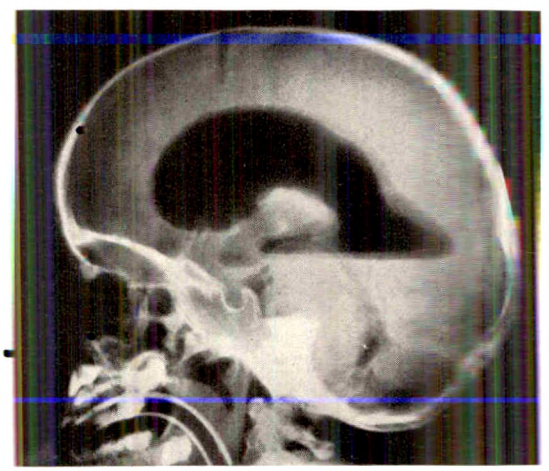


FIG. 2. Case I. There is a marked dilatation of the entire ventricular system with some flattening of the pontine cisterns and no air in the subarachnoid space above the interpeduncular cistern. The thickness of the pons is 3.6 cm. The floor of the fourth ventricle is convex; the curve of the aqueduct is less acute than normal, and there is an upward bulging of the posterior portion of the floor of the third ventricle.

The parents of the second child (Case II) refused to accept a fatal prognosis and took the child to another hospital where he was operated upon and died a few months later. The pathological report of a biopsy was pontine glioma. No postmortem examination was made in this case.

The pneumo-encephalographic appear-

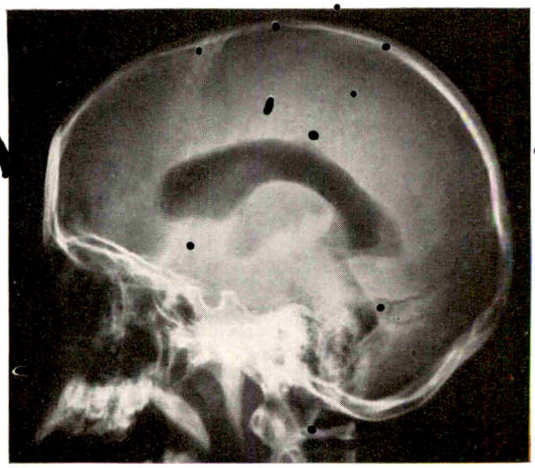


FIG. 3. Case II. There is a moderate dilatation of the entire ventricular system with no air in the subarachnoid space. The thickness of the pons is 4.2 cm. The pontine cisterns are obliterated; the floor of the fourth ventricle is convex; the curve of the aqueduct is less acute than normal; and the tumor has caused a slight bulging of the posterior portion of the floor of the third ventricle.

ance of pontine tumors has been described by Lysholm,[1,2] but we feel the films in these 2 cases are so characteristic that they should be reported.

In the normal encephalogram the thickness of the pons, including the basilar and tegmental portion from the roof of the cisterna pontis to the floor of the fourth ventricle, measures about 2.5 to 3.0 cm., as reported by Davidoff and Dyke.[3] However, this is a variable figure depending upon the distance at which the exposures are made.

In cases of glioma of the pons, if the ventricular system is well outlined by air, we feel the pneumo-encephalographic appearance is unmistakable. Both of these cases showed a tumefaction of the pons which was well outlined between the floor of the fourth ventricle and the pontine cisterns. These tumors produced a rounded filling defect in the floor of the posterior portion of the third ventricle and a posterior bulging of the floor of the fourth ventricle and aqueduct. There was no air in the subarachnoid space over the cerebrum, and there was flattening or obliteration of the pontine cisterns.



FIG. 4. A comparison of the specimen in Case I (*A*) *above*, with a normal brain (*B*) *below*.

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VOL. 30, NO. 6

LESIONS OF THE ACROMIOCLAVICULAR JOINT CAUSING PAIN AND DISABILITY OF THE SHOULDER

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AMONG the various lesions which may induce persistent or recurrent pain and disability of the shoulder, those involving the acromioclavicular joint have received little attention, although they seem to be common.

The acromioclavicular joint has an important function. The range of motion of the arm depends significantly on the mobility of the scapula. The mobility of the scapula depends in turn on the mobility of the shoulder girdle in the sternoclavicular and acromioclavicular joints. These joints render the connection between scapula and sternum sufficiently flexible to permit fairly extensive motion of the scapula along the wall of the chest. Roentgenograms show that there is motion in the acromioclavicular joint during all movements of the arm except pronation and supination (Fig. 1). This means that diminished mobility of the acromioclavicular joint will interfere with most of the movements of the arm in the shoulder.

METHOD

The acromioclavicular joint space is approximately at a right angle to the wing of the scapula. The position of the scapula is variable. In persons with a flat chest and a straight thoracic spine, the shoulder blade is almost vertical and nearly parallel to the frontal plane. The deeper and rounder the thoracic cage, the greater the angle which the scapula forms with the frontal plane; the rounder the curve of the thoracic spine, the greater the deviation of the scapula from the vertical position. The roentgenographic projection is determined, therefore, not by the position of the patient, but by the position of his or her scapula. The central ray should be perpendicular to the wing of the scapula at the level of the acromion. This may be achieved either by placing the patient supine with the shoulder blade of the involved side flat on the cassette or table, the other shoulder being raised accordingly, or by having the patient stand upright with his back to the film and his shoulder drawn backward like a soldier standing at attention. In adipose patients and in the presence of a pathologic or increased physiologic kyphosis, the scapula remains inclined, with its inferior margin pointing backward even in the "attention" position. It is necessary, therefore, to tilt the tube so that the central ray points upward in these cases. The appropriate angle can be determined by palpation of the superior surface of the acromion, the articular surfaces standing usually in the same direction, with the joint space at a right angle to the surface of the acromion. Axial projections, made through the axilla either cephalocaudad or caudocephalad, may give additional information; but their value is somewhat limited as the head of the humerus, parts of the coracoid process, and the glenoid cavity are usually superimposed upon the acromioclavicular joint. Moreover, in the presence of a painful and disabled shoulder, the arm cannot always be abducted sufficiently to obtain a satisfactory projection.

With the exposure factors applied for the ordinary anteroposterior roentgenograms of the shoulder, the acromioclavicular joint usually becomes moderately overexposed or overpenetrated, but not so much as to preclude interpretation, especially if a spot light is used for viewing the films. If this roentgenogram suggests that a lesion of the acromioclavicular joint is responsible for the complaint, additional exposures with a

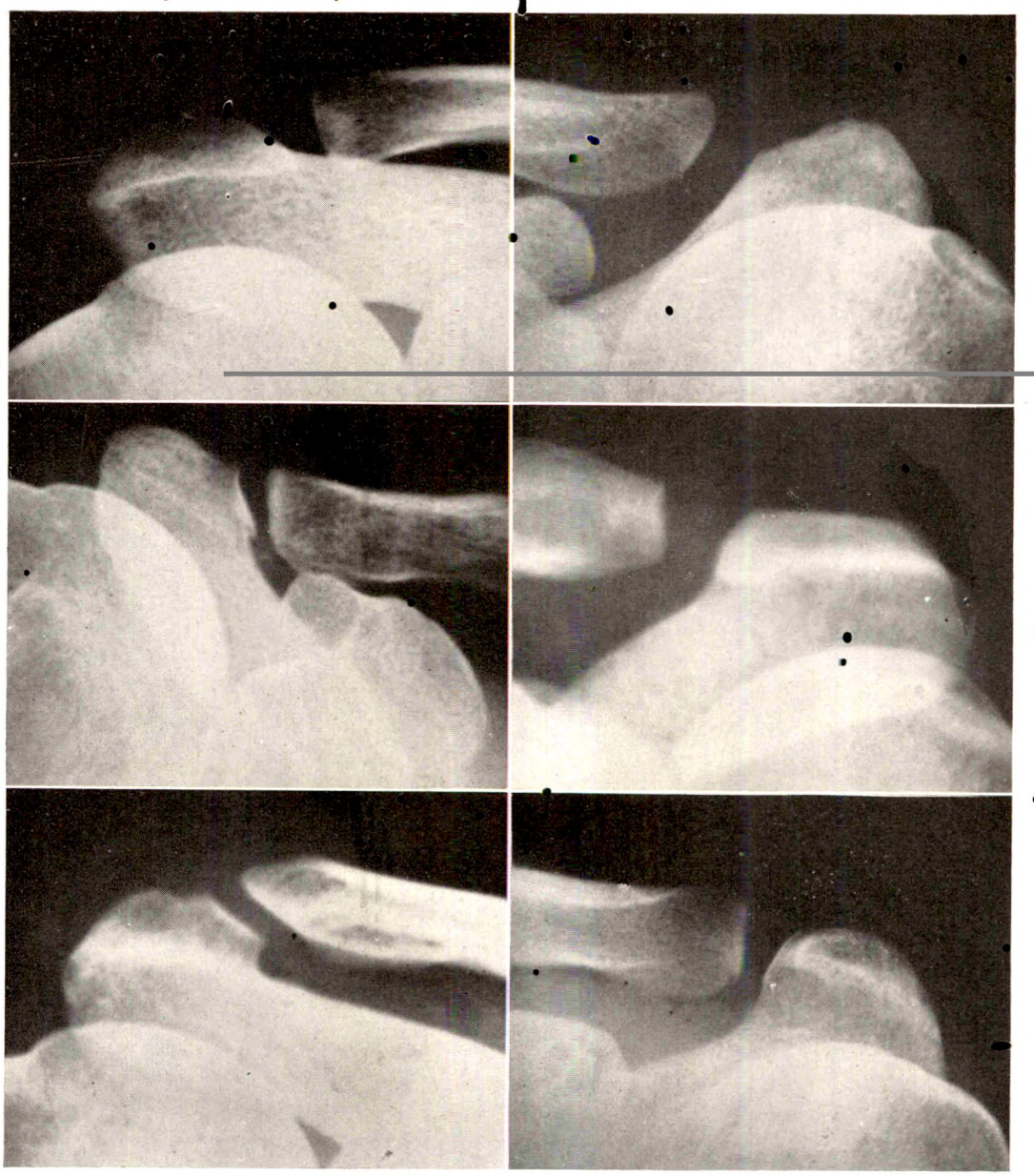


FIG. 1. Normal acromioclavicular joints. Note the variability of outlines, position, and width of joint spaces. Anteroposterior projection, as described in text.

lower kilovoltage may be made. Roentgenograms made at a 36 in. distance, with 30 to 35 kv. and 50 ma-sec., using par-speed screens, are satisfactory. A Potter-Bucky grid need not be used.

NORMAL APPEARANCES

The joint space of the acromioclavicular joint varies between 2 and 5 mm. in width, as measured on the roentgenogram taken at a 36 in. distance, and is wider in the marginal than in the central areas, since the articular surfaces are usually convex as seen from above. An unusually wide joint space may indicate the presence of an articular disc, which is sometimes found as a normal variation, according to anatomic texts.[5] The articular bony surfaces are

smooth and clean-cut; seen edgewise, they may be straight, notched, concave, or convex, and may differ in shape in the two joints of the same person. The capsule is clearly visible as a cylindric shadow, connecting the bony ends and raised very slightly, if at all, above their level in the region of the joint space. The articular bones have a fine mesh-like structure with a very thin cortical layer. Supernumerary ossicles arising from secondary centers of ossification, which are common in the region of the sternoclavicular joint, do not seem to have been observed in the acromioclavicular joint.[7]

The clavicle articulates with the acromion close to the anterior edge of the medial surface of the acromion.

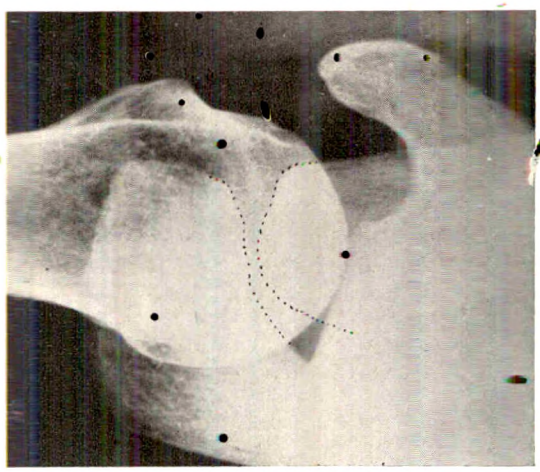


FIG. 2. Normal acromioclavicular joint, cephalocaudal projection upon a curved cassette placed in the axilla. The outlines of the joint, redrawn, are covered by the head of the humerus in this position.

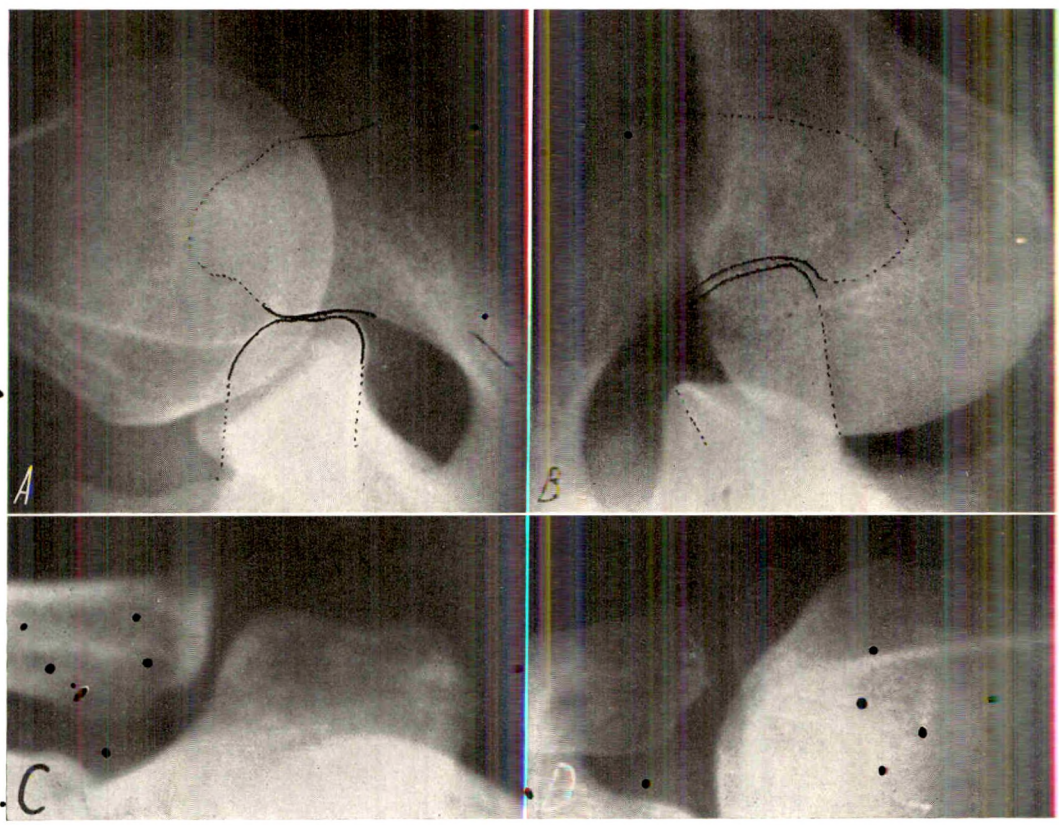


FIG. 3. Position of the articular surfaces during forward (*A*) and backward (*B*) motion of the arm (redrawn) and during normal position (*C*) and elevation (*D*) of the arm.

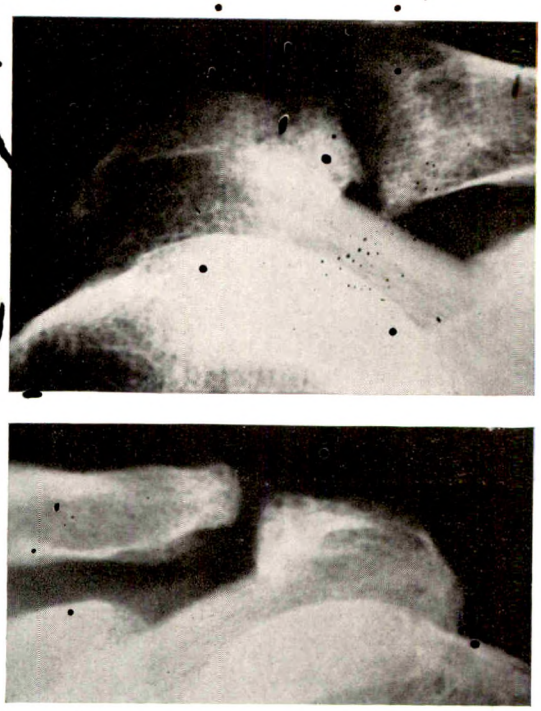


FIG. 4. Changes resulting from recent trauma of the shoulder. Note stippled rarefaction of the bones and fuzzy outlines of articular surfaces.

FRACTURES AND DISLOCATIONS

Fractures of the acromion and dislocations of the acromioclavicular joint are mentioned in most textbooks. They are usually the result of direct trauma. Fractures seem to occur mainly in the lateral part of the acromion, wherefore they rarely involve the joint. As a rule, displacement of fragments is only slight, and the fracture heals readily with a callus formation giving the acromion a dense and rough appearance. Sometimes the fracture line is very difficult to recognize, and a large number of views made in different projections is necessary to demonstrate it. Since the clinical manifestations are usually very mild, it is quite possible that some of these fractures escape recognition. Dislocations of the acromioclavicular joint are less common than fractures, the ratio being 1:4 in our observations. Usually the clavicle is dislocated upward, with or without overriding of the clavicle. Downward dislocation is rare.[1] The coracoacromial and acromioclavicular liga-

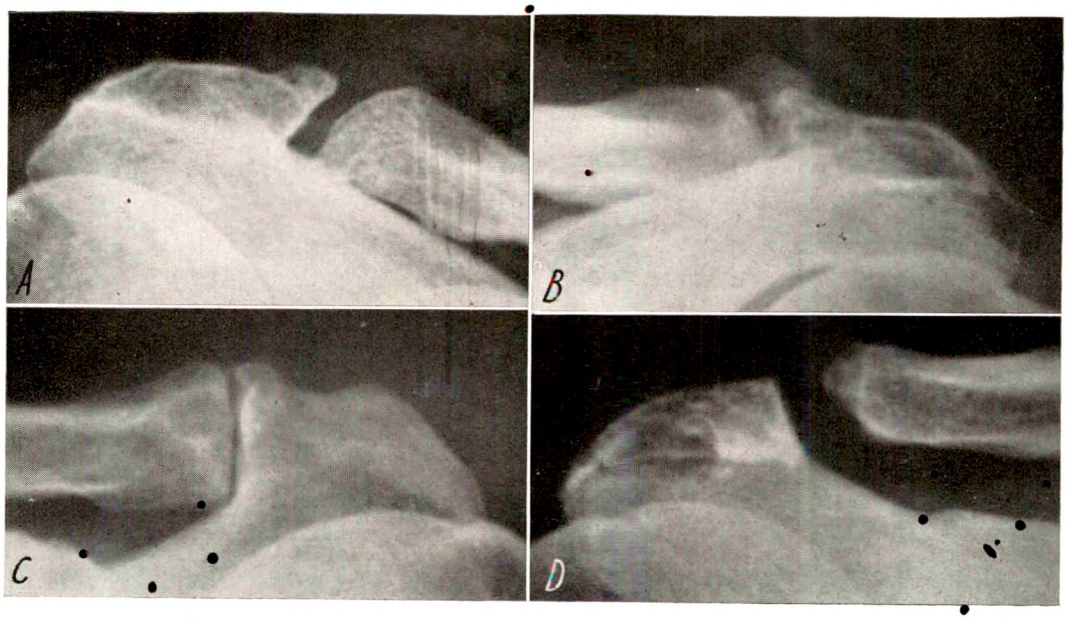


FIG. 5. Hypertrophic (osteo-) arthritis. *A*, normal side; *B*, involved side. *C*, a more advanced case, with severe narrowing of joint space. *D*, another case during an acute exacerbation of symptoms, with widening of joint space possibly caused by effusion.

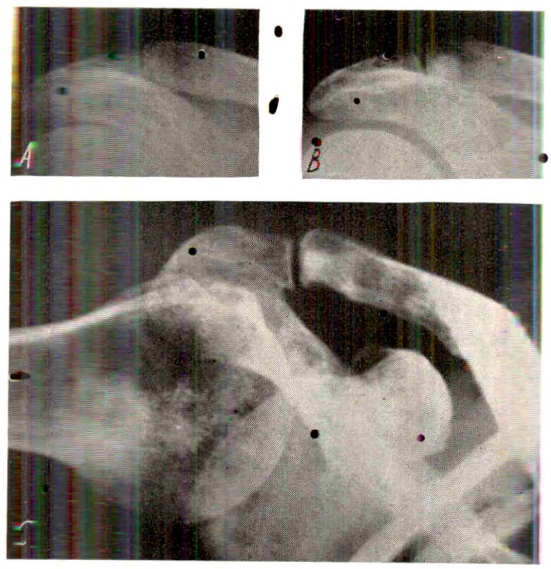


FIG. 7. Tumor metastasis. A patient with metastasis of cancer of the breast. *A*, joint normal; *B*, three months later, a small metastasis has lodged close to the articular surface of the clavicle. *C*, five months later, an osteophyte has formed, simulating osteo-arthritis.

cipient and early phases. Later, the capsule becomes enlarged and the articular bone rarefied. The joint space grows narrow, and the articular surfaces may appear eroded. Complete bony ankylosis may develop. In other cases, the lesion becomes arrested before ankylosis occurs, and the articular surfaces with the subchondral bone may then show areas of increased density scattered between areas of rarefaction. Sometimes the articular surfaces become severely eburnated. At this stage of the lesion, rheumatoid arthritis is difficult to distinguish from osteo-arthritis.

Osteo-arthritis (hypertrophic arthritis, degenerative joint disease) seems to develop in the acromioclavicular joint as a result of occupational strain or of repeated trauma. A distinction between "traumatic" and "degenerative" osteo-arthritis is not made here, as the two lesions are anatomically identical. Osteo-arthritis is the most common lesion of the acromioclavicular joint, its incidence being about twice as high as that of all other lesions combined. It produces definite disability of the shoulder. Roentgenograms show the capsule definitely enlarged, usually with moderate widening of the joint space, perhaps as a result of effusion. At more advanced stages, the articular surfaces are eburnated and rough, and their margins may show bony overgrowth; the joint space may then grow narrow. The subchondral bone is usually coarse, the bone trabeculae being increased in thickness but diminished in number. Sometimes small areas of rarefaction are scattered through the area of increased density.

TUMORS

Metastases of cancer of the breast, bronchial carcinoma, and other tumors, are occasionally found lodged very close to the acromioclavicular joint space both in the clavicular and in the sternal articular bones. In 3 of these instances a gradual, apparently reactive eburnation and overgrowth of the articular surfaces was observed in the absence of clinical signs and symptoms. Primary new growth arising from the acromioclavicular joint was not recorded.

CLINICAL MANIFESTATIONS

Dislocations are usually recognized on physical examination, although their recognition is not always easy. Fractures of the acromion are more difficult to diagnose, since pain and disability are very slight in most of these cases, as above mentioned.

Irrespective of its kind and stage, arthritis of the acromioclavicular joint causes pain in the shoulder, often radiating into the arm, wrist, and fingers, with definite limitation of the movements involved in bringing the arm above shoulder level, across the chest, and onto the back. Pronation and supination are not limited, unless pain is so severe as to produce a reflex inhibition; this distinguishes acromioclavicular from humeroscapular arthritis. But the other signs and symptoms are not characteristic, as they may be produced by bursitis, myositis, traumatic lesions of the muscles, tendons, and bones of the shoulder and radicular neuritis caused by disease of the cervical spine. A point of tenderness

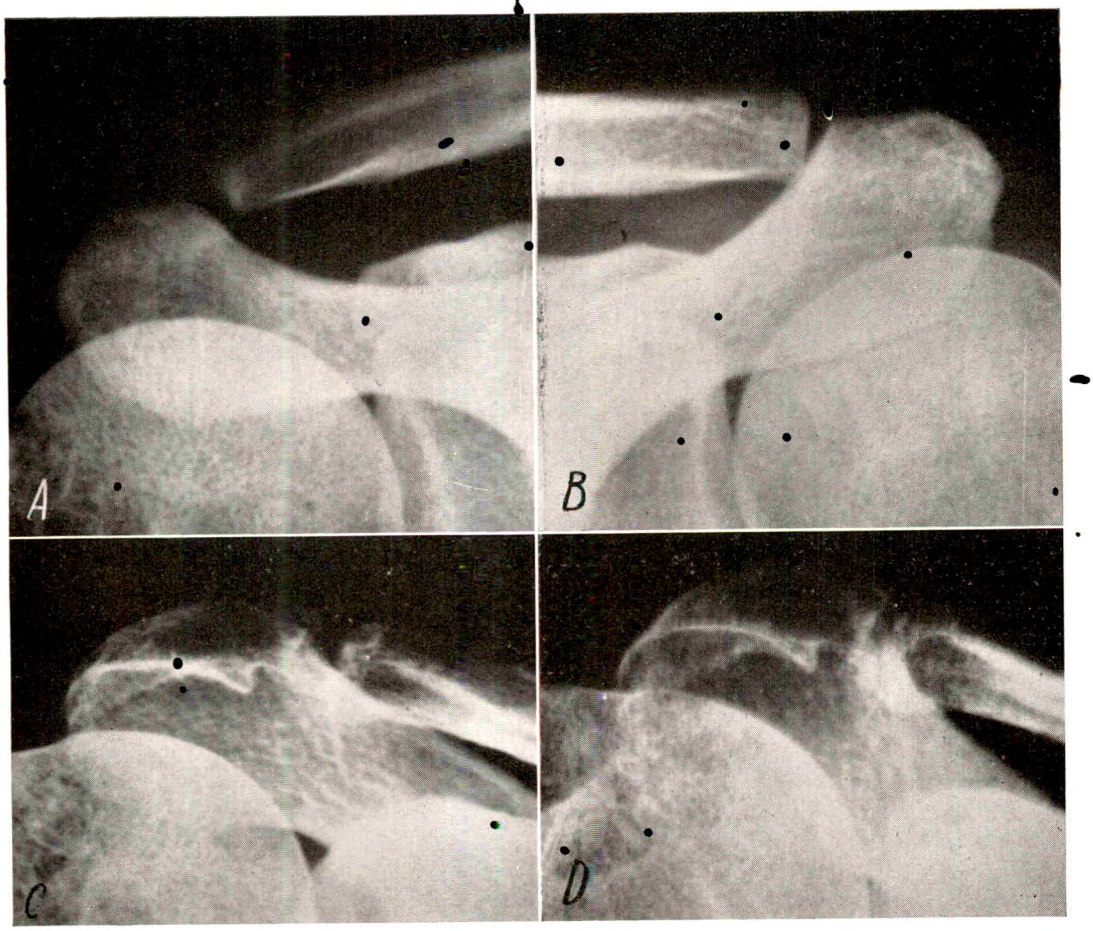


FIG. 6. Atrophic (rheumatoid) arthritis. *A*, normal side; *B*, involved side of a patient with atrophic arthritis of the hands, elbows, and lumbar spine. *C* and *D*, development of ankylosis as a result of rheumatoid arthritis; *C* was taken sixty-four days after *D*.

ments may become injured, and the joint capsule may be torn.

ARTHRITIS

As a synovial or diarthrodial articulation, the acromioclavicular joint may become the seat of any of the various kinds of arthritis. Tuberculous arthritis has been recorded.[4,8] Gonorrheal arthritis leading to complete bony ankylosis was observed in one patient whose roentgenograms are not available. Trophic changes, marked by irregular stippled rarefaction of the articular and para-articular bone, were observed in 2 cases of severe brachial neuritis.

Acute arthritis, clinically indistinguishable from subacromial bursitis, develops occasionally either as a result of sprain, or as a sequela of infection of the nasopharynx. In these cases, the shadow cast by the joint capsule is definitely enlarged (Fig. 5*A*), but there are no other changes seen on roentgenograms. The lesion seems to heal spontaneously in the course of several weeks, and the capsule regains normal size.

Rheumatoid (atrophic) arthritis of the acromioclavicular joint sometimes develops in patients with rheumatoid arthritis of other joints, including those of the spine. Like the peripheral joints, the acromioclavicular joint does not show definite changes on roentgenograms during the in-

confined to the acromioclavicular joint may suggest the presence of a lesion of this joint; but, as a rule, the whole shoulder is tender on palpation and pressure irrespective of the localization of the lesion. Atrophy of a group of muscles may develop, as in the presence of other diseases which induce the "painful shoulder syndrome." The diagnosis is determined by the roentgen findings in a majority of cases.

CONTROL SERIES

In a series of 50 persons without any pain or disability of the shoulder, of various age groups including children and subjects over seventy years of age, calcified subacromial and subdeltoid bursae were present in 3 instances, and various lesions of the cervical spine in 7 cases; the acromioclavicular joints were normal in all of them. Conversely, in a series of 50 cases of recurrent pain and disability of the shoulder, the acromioclavicular joints were found involved alone in 9 patients, and together with flattening of cervical intervertebral discs in 2 instances. This would seem to suggest that the lesions of the acromioclavicular joint here reviewed are actually responsible for the clinical manifestations described, and are not accidental and irrelevant findings.

INCIDENCE

During the past eleven months, when special attention was paid to acromioclavicular lesions, 28 cases were observed. A review of roentgenograms of the shoulder made before that time seems to show that this incidence is the usual one. Acromioclavicular lesions were about eight times as frequent as humeroscapular arthritis; three times as frequent as subdeltoid and subacromial bursitis combined; nearly as frequent as the various traumatic lesions of the muscles and tendons of the shoulder girdle in an industrial and agricultural district; and about one-half as frequent as radicular neuralgia caused by disease of the cervical spine. A study of the age and sex distribution did not disclose any significant findings.

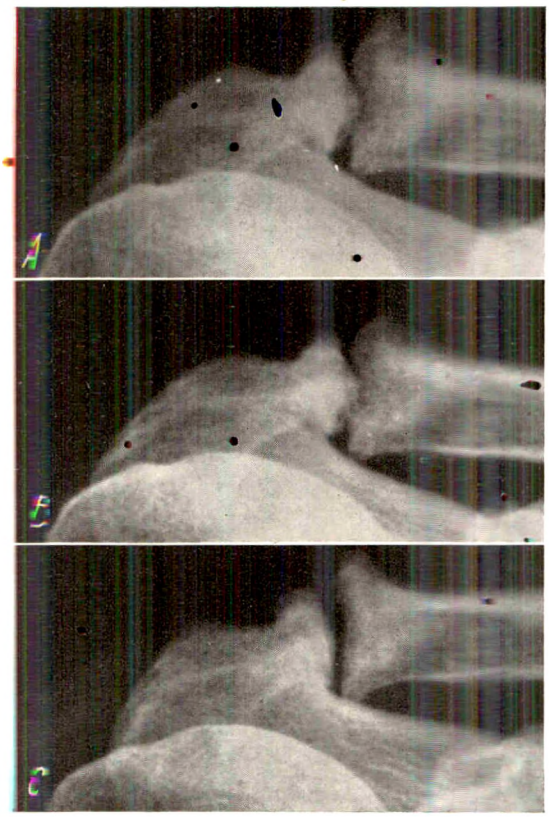


FIG. 8. Result of roentgen therapy. Osteo-arthritis, with narrowing of joint space and marginal overgrowth. *A*, appearance in September, 1942; *B*, May, 1943. No form of treatment was given during the interval. *C*, June, 1943, three weeks after *B* was taken; three treatments with 50 r each were given during the interval. No other therapy was applied.

TREATMENT

All of the patients were referred to this department because various modes of therapy, such as heat applications, analgesics, splints, and injections of a local anesthetic into the deltoid region had failed to produce appreciable or permanent relief. Short wave therapy was ineffective.

Roentgen therapy was applied, using 140 kv., 20 ma., 0.5 mm. Cu plus 1 mm. Al filtration, 50 cm. target-skin distance, with doses of 50 to 70 r (measured in air), through fields 5 cm. square, directed to the acromion from anteriorly or cephalad, at

intervals of four to five days. The results were tested by the method recently described.* In some of the patients pain began to subside rapidly after the second irradiation; in others, six to eight treatments were required to bring about complete or almost complete relief. The movements of the arm in the shoulder usually became normal about ten days after disappearance of the pain; but some slight limitation, generally unnoticed by the patients, tended to persist for several months. A definite decrease of the capsular enlargement was noted on roentgenograms in all instances, and a re-widening of a narrowed joint space, together with decrease of the erosion of the articular surfaces, was seen in 2 cases. Of the 11 patients treated, 5 have been free of symptoms for over six months. No ill effects of this treatment have been recorded.

SUMMARY

1. Pain and limitation of movements of the shoulder are often induced by lesions of the acromioclavicular joint.

2. Osteo-arthritis seems to be the most common lesion which involves the acromioclavicular joint.

3. The clinical manifestations of the various acromioclavicular lesions are virtually identical with those caused by bursitis, myalgia, radicular neuralgia, and traumatic injury of the shoulder girdle.

4. The roentgenographic method and the significant roentgen findings are described.

5. The lesions respond favorably to roentgen therapy with small doses.

* Oppenheimer, A. Development, clinical manifestations and treatment of rheumatoid arthritis of the apophyseal intervertebral joints. AM. J. ROENTGENOL. & RAD. THERAPY, 1943, *49*, 49–76 (see page 73).

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* I am greatly indebted to Miss Florence E. Turner, Librarian of Boston University School of Medicine, for her assistance in finding these references.

VOL. 51, NO. 6

TRAUMATIC SEPARATION OF THE UPPER FEMORAL EPIPHYSIS

A BIRTH INJURY*

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TRAUMATIC separation of the upper femoral epiphysis at birth is a rare and interesting lesion. Pfeiffer, in 1936, listed only 15 reported cases, including his own,—12 from the German and 3 from the Italian literature. He was not aware of 3 American cases already published. To these can now be added 3 more but, even including some reports unknown or unavailable to us, the total number would hardly exceed 30. Birth fractures through the diaphyses of clavicle, humerus and femur are common and well known. Separation at birth of the humeral epiphyses and the lower femoral epiphysis is less frequent but has often been described. This is not true, however, of separation of the *upper* femoral epiphysis. In the last ten years neither the Lying-In Hospital nor the Woman's Hospital (both in New York City) have a record of such a birth injury. Moreover, roentgen examination is essential in its diagnosis, as will be apparent in the following case report:

The patient, A. B., a baby girl, was born at term May 16, 1942, in another hospital. A transcript of the record from that institution notes that the position of the fetus was persistently occipitoposterior. Low forceps were applied after thirteen hours of labor. Rotation could not be accomplished and the head was delivered in the posterior position. No difficulty or unusual manipulation is known to have occurred in the extraction of the legs. It is possible, however, that after this operative delivery resuscitation was necessary and the baby's legs may have been abnormally or forcibly handled during this procedure.

Examination of the infant immediately after birth revealed no abnormalities, except of the lower extremities. The thighs were abducted, the knees flexed and the feet deformed (bilateral clubfoot). Each leg was apparently paralyzed and the reflexes were absent. The Wassermann reaction of the mother's blood and the umbilical cord blood was in each case negative.

A copy of the roentgen report dated May 19 states that the bony structure of the lower extremities "is relatively normal in appearance. No dislocation of the hips" and mentions considerable atrophy of the soft tissues. On May 28, twelve days after birth, the report describes "a streak of increased density along the shaft of the left femur in the trochanteric region . . . probably a periosteal reaction."

The infant was admitted to the Orthopedic Service of St. Luke's Hospital on June 4, nineteen days after birth. There was a striking frog-like deformity of the hips, caused by marked flexion, abduction and external rotation. The thighs and hips were swollen, indurated and discolored bluish-red. There was marked limitation of motion at the hip joints and also in the knees and ankles. It was impossible to bring the thighs into a normal position or to extend the legs at the knees. On each side was a talipes equinovarus deformity. Neurological examination revealed normal reflexes in the head and arms but no reflexes in the legs. The skin of the feet and shins seemed unusually shiny, suggesting trophic changes. The baby constantly dribbled urine and this possible indication of a neurological bladder, coupled with the absent reflexes and the skin changes, led to a tentative diagnosis of spastic paraplegia, associated with a spina bifida or spinal birth injury. No manifest spina bifida, however, could be found on physical examination or later by roentgenography. The Kline test was negative.

The diagnosis was clarified on viewing the roentgenograms taken on June 4, the day of admission (Fig. 1). The upper end of each femur was displaced outward and upward from its usual position in relation to the acetabulum. A large mass of heavy callus, quite dense and not fluffy, surrounded the upper end of the right femur. There was a well marked subperiosteal reaction (new bone formation beneath the

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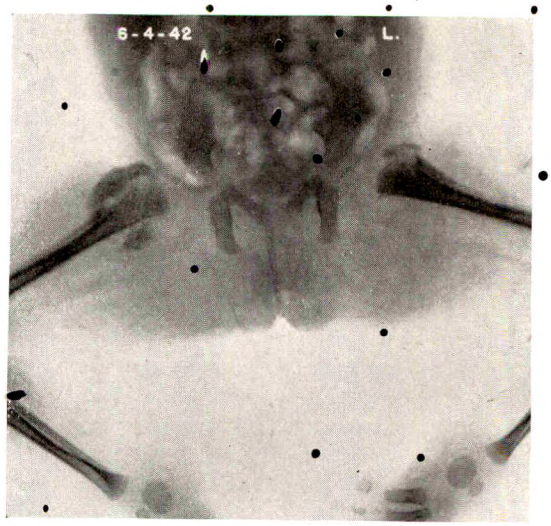


FIG. 1. Roentgenogram taken on admission, nineteen days after birth. Note the heavy callus and prominent subperiosteal reaction around the proximal end of each shaft, which is displaced upward and outward. The hips are held in a frog-like position.

raised-up periosteum) along the proximal two-thirds of the lateral aspect of the femoral shaft, and a fainter, narrower strip along the whole medial margin. The reaction was widest and most prominent in the region of the callus. At the upper end of the left femur was situated a smaller mass of callus, mainly on the upper and lateral aspect. From this a wide, very dense zone of subperiosteal reaction extended down along the lateral edge of the shaft to about the mid-point and apparently continued to the distal end, though visible only along the medial border. No fracture of either femoral diaphysis was seen.

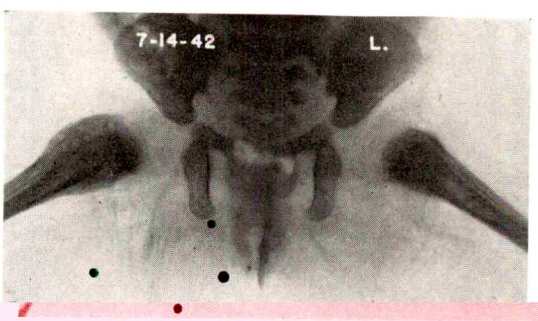


FIG. 2. Roentgenogram two months postpartum. The callus masses have undergone considerable absorption and organization. Note the wide zone of subperiosteal new bone formation along each shaft.

Roentgenograms taken at later dates show progressive organization and absorption of the callus. On July 14, two months postpartum, there is still the frog-like position of the legs (Fig. 2). The space between each acetabulum and the upper end of the femur is still widened. The callus is now organized into new bone and forms a compact, smoothly outlined mass surrounding the proximal end of each diaphysis, the original outlines of which are still discernible within it. A wide zone of dense, laminated, subperiosteal new bone formation extends down the lateral border of each femoral shaft to the distal end.

On August 25, a little over three months post-

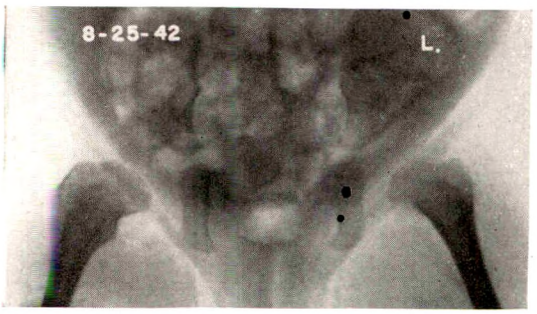


FIG. 3. Roentgenogram about three months postpartum, legs forcibly adducted. Further absorption of callus and subperiosteal new bone; reconstruction of upper end of each femur.

partum, this periosteal reaction is much less prominent (Fig. 3). The club-like enlargement at the proximal end of each diaphysis is smaller and considerably remodeled. In the abducted position the space between acetabulum and femur is still widened; in forcible adduction this distance is not so great. The upper end of the shaft in either position is higher than normal in relation to the Y-shaped fissure of the acetabulum

On October 9 (Fig. 4 and 5), about five months after birth, the evidences of previous injury are almost gone. The proximal end of each femur, through absorption and remodeling and through the growth process of ossification of the neck, has an appearance more like the normal. Abduction is still maintained and it is impossible to bring the legs together. There is still outward and upward displacement, as will be seen on comparison with roentgenograms of a normal five months old child (Fig. 6). The nucleus of each capital epiphysis is ossified.

During this period corrective measures were

applied to the congenital equinovarus deformities of the feet but no treatment of the hips was instituted.

LITERATURE

The paucity of reports in the literature, especially in American or English journals, prompts a rather complete résumé of them. Pfeiffer states that Pforte[12] described the first case in 1908 but Ehrenfest[3] alludes, without further particulars, to 2 cases reported by Foersterling in 1898. There is also an intriguing will-o'-the-wisp reference in Poland's large monograph, published in 1898: Poland[13] quotes Simpson who

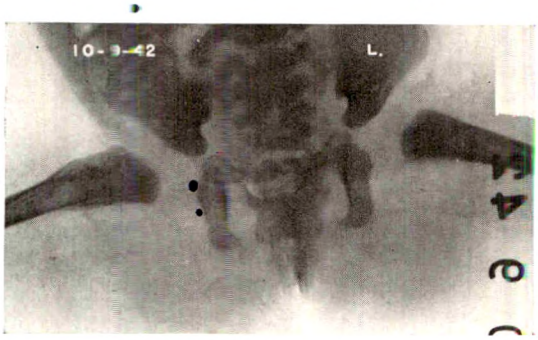


FIG. 4. Roentgenogram about five months postpartum. Hips still flexed, abducted and externally rotated. Remodeling process well advanced. At the proximal end of the right femur the original outlines of the shaft are still visible within the organized callus.

quotes Küstner who quotes Thudichum (1855) as knowing of a case of separation of the upper femoral epiphysis, apparently reported by a man named Bertrandi! Nathan's case[10] and Puppel's[15] case were described as examples of traumatic dislocation of the hip at birth. Harrenstein[6] and Camera[1] independently and almost simultaneously reported their series of observations from which they correctly appraised the true nature of the injury and the resultant lesion. Kleine,[8] in 1933, disproved the contention of Nathan and Puppel and showed that their cases, along with his, were instances of separation of the epiphysis. It remained for Pfeiffer,[11] in 1936, to review and summarize the previous articles, list the cases known to him,

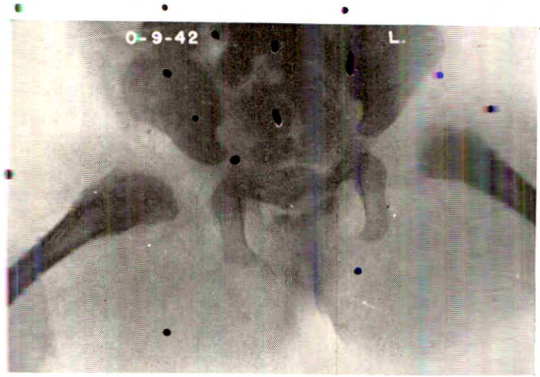


FIG. 5. Roentgenogram about five months postpartum, legs slightly adducted. Note that the proximal end of each femur is still displaced upward and outward. The osseous nucleus of each femoral head is faintly visible and correctly placed in the acetabulum.

and add 3 of his own. Since then Meier[9] has contributed 2 more examples (1939).

In the United States, on the other hand, Truesdell reported a case as early as 1918. This author, who has had a wide experience with birth fractures and epiphyseal injuries, correctly described the lesion and published good roentgenographic studies of it.[19] In 1935, Snedecor, Knapp and Wilson,[17]

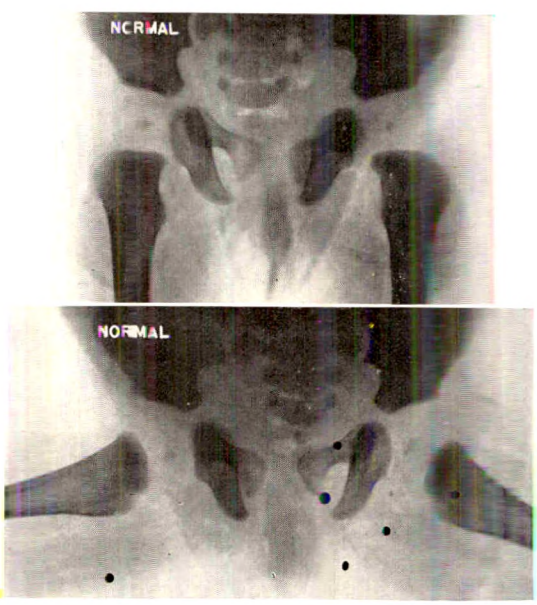


FIG. 6. Roentgenograms of the hips in a normal child aged five months. *Above*, in usual position. *Below*, hips in flexion, abduction and external rotation.

who probably did not have access to Truesdell's paper, reported 2 instances of birth injury to the upper end of the femur to which they gave the name "traumatic ossifying periostitis of the newborn." The roentgenograms of their cases, together

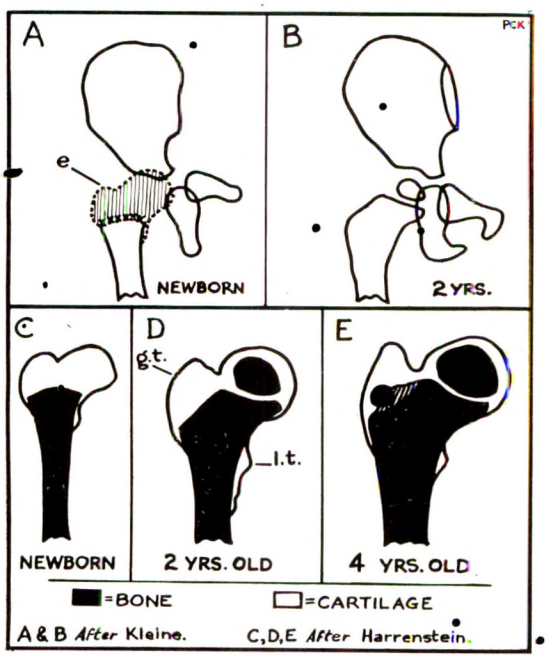


FIG. 7. Diagrams showing development of upper end of femur.

A, at birth. Shaded structure (*e*) is the large cartilaginous upper femoral epiphysis.

B, osseous structures present at the age of two years.

C, at birth the femoral head and both trochanters are included in the cartilaginous epiphysis.

D, at two years of age the head and trochanters (*g.t.* and *l.t.*) are considerably separated through the growth of the femoral neck.

E, at four years, the separation is complete. The head and greater trochanter each have a bony nucleus and separate growth disc.

with the histories and end-results, show that they really are examples of traumatic separation of the upper femoral epiphysis.

There are also a few articles[2,14,16] which we have been unable to obtain but their titles suggest additional data on this subject. Two probable cases are listed in Thorndike and Pierce's[18] large chart (H.K.P. and R.G.) but only fragmentary details are given. Undoubtedly several other instances of this injury have been recognized by keen observers but not published. Truesdell,[21] for instance, knows of at least 3 typical cases besides the one in his paper.

Brief accounts of many of the cases in the literature follow (see also Table 1 and Fig. 8):

CASES FROM THE LITERATURE

Truesdell (1918): Breech presentation, delivered by breech extraction. Great difficulty in delivery of shoulders and after-coming head, necessitating vigorous manipulation of lower part of infant's body. At birth, discovery that baby did not move one leg. In a few days, large and firm swelling of thigh. One month later, swelling diminished and function largely restored. Three months after injury, free motion at hip but bony thickening felt in upper thigh. No treatment.

Roentgenograms (Fig. 8, *A*, *B*, *C*): Two weeks postpartum—upper end of femur displaced laterally from acetabulum; large, fluffy callus formation around upper end of shaft. Four weeks postpartum—still lateral displacement; callus denser; subperiosteal new bone formation beneath stripped-up periosteum along proximal two-thirds of shaft. Three months postpartum—still lateral displacement; callus now organized and large mass of new bone surrounding upper end of shaft and region of lesser trochanter; marked subperiosteal new bone formation; ossific nucleus of femoral head already present on affected side but absent on the other; accelerated growth of affected femoral neck.

Nathan (1928) [From Kleine]: Version and breech extraction. During prolonged delivery it was necessary to pull strongly on left leg. At birth, this leg swollen, slightly shortened, externally rotated, abducted, and immobile. First seen by Nathan three and one-half months after injury; roentgenogram at this time showed femoral shaft displaced upward and upper third of shaft bowed outward (varus-like). Treated for eight weeks by "reposition"—plaster immobilization in mid-abduction and strong internal rotation. One and one-half years after birth, position of left femur normal on roentgenograms. Nathan considered this a case of traumatic dislocation of hip but later authors (Kleine, Pfeffer) disagree with him.

Harrenstein (1929): Case 1. Version and breech extraction. Difficult delivery, including unsuccessful strong traction on right leg. After birth, doughy swelling and no spontaneous movement in right hip; upper femur displaced laterally but leg held in adduction. Pain on passive motion. Three weeks' treatment in a form of extension with vertical traction and occasional lateral pressure on the femoral shaft. After roentgen examination showed improvement treatment was continued for three more weeks. One and

TABLE I

Case	Author	Year of Report	Method of Delivery	Days Postpartum, First Examination	Time Postpartum, Final Examination	End-Result at Time of Last Examination	Treatment
1	Pforte	1908	Version and extraction	2	28 days + autopsy		
2	Truesdell*	1918	Breech extraction	14	3 mo.	Organized callus; free motion at hip	None described
3	Winter	1927	Version	3	Unknown + autopsy		
4	Nathan	1928	Version and extraction	60	20 mo.	Good	"Reposition"; plaster immobilization in mid-abduction and internal rotation
5	Harrenstein, Case I	1929	Version and extraction	17	19 mo.	Good shape and function	Extension, vertical traction + lateral pressure on shaft
6	Harrenstein, Case II	1929	Version and extraction	15	11 mo.	Coxa vara	None
7	Camera, Case I	1930	Version?—breech extraction	2–3	2 yr.		
8	Camera, Case II	1930	Version?—breech extraction	2–3	2 yr.	Severe coxa vara	
9	Camera, Case III	1930	Version?—breech extraction	17	Unknown		
10	Puppel	1930	Version and extraction	5	9 mo.	Return to normal	Abduction
11	Kleine	1933	Version and extraction	4	7 mo.	Severe coxa vara	Supporting fixation bandage; abduction, slight internal rotation
12	Med. Clinic	1935	Version and extraction	5	Unknown		
13	Snedecor,* Case I	1935	Version and extraction	40	4 mo.	Callus organized	None
14	Snedecor,* Case II	1935	Version and extraction	5	24 days	Large callus	None
15	Pfeiffer, Case I	1936	Version and extraction	14	4 wk. + autopsy		None
16	Pfeiffer, Case II	1936	Version and extraction	3	14 mo.	Return to normal; no coxa vara	Lorenz primary position
17	Pfeiffer, Case III	1936	Version and extraction	42	7 mo.	No coxa vara	Tibial extension traction
18	Epstein and Klein	1936	Version and extraction	?	?		
19	Meier,* Case I	1939	Breech presentation and extraction	10	1 yr.	No coxa vara	Extension; 90° flexion at hip; internal rotation
20	Meier,* Case II	1939	Version and extraction	?60	8 wk. + autopsy	Mild coxa vara	None
21	Kennedy*	1944	Head presentation low forceps	14	5 mo.	Poor; still abducted and externally rotated	None yet

* New cases added to Pfeiffer's list.

one-half years after injury, shape and motion of hip normal.

Roentgenograms: Seventeen days postpartum—right femoral shaft displaced upward and outward; upper limit of diaphysis less sharply defined than on opposite side; fluffy callus on medial aspect of upper end of femur. Five weeks postpartum—less displacement; callus compact and spread out over upper end. One and one-half years postpartum—position good; ossific nucleus for femoral head larger than on uninjured side; cartilaginous disc between head and neck narrower than on uninjured side.

Case II. Version and breech extraction. Difficult delivery, with unsuccessful strong traction on right leg in order to disengage foot wedged between fetal head and maternal pelvis. At birth, hip swollen and immobile, continuing thus for four to five weeks. Expectant treatment at home for various reasons. Partial return of active motion at end of seven weeks but full range not till eleven months (even then, slightly limited extension).

Roentgenograms (Fig. 8, *D*, *E*): Seventeen days postpartum—upward and lateral displacement of proximal end of femoral shaft; upper limit of diaphysis fuzzy and surrounded by callus. Seven weeks postpartum—callus increased but compact; subperiosteal new bone formation along upper third of shaft. Eight months postpartum—femoral head nucleus visible on affected side but not on other. Eleven months postpartum—still slight lateral displacement; head nucleus larger and growth disc narrower on affected side; femoral neck and medial border of upper shaft curved on injured side, indicating coxa vara deformity; diaphysis shortened 0.5 cm.

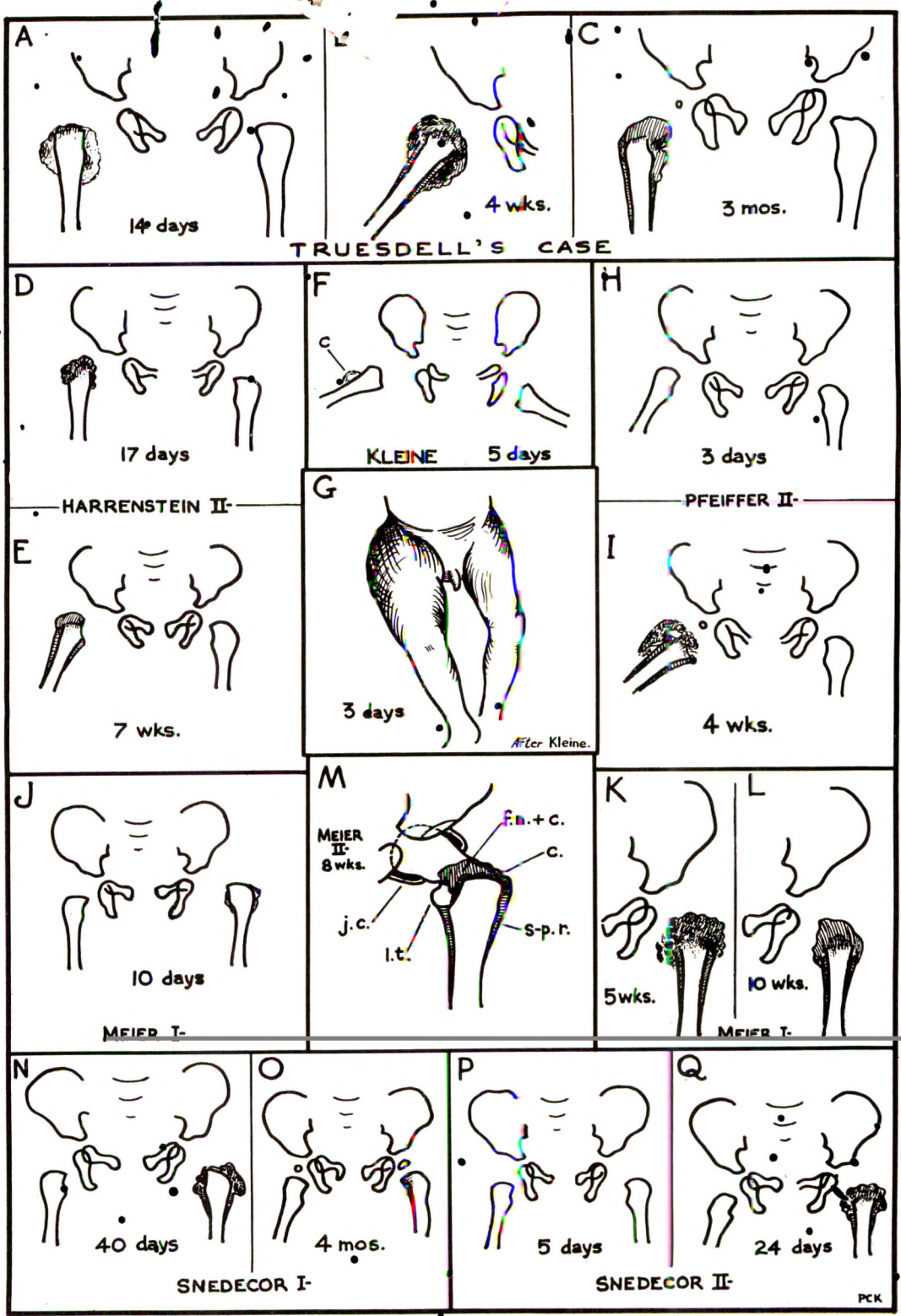


FIG. 8. See next page for description.

Puppel (1930) [From Kleine]: Version and breech extraction. Traction on right foot. On fourth day after birth, swelling, shortening, external rotation and limited motion of right thigh. Treatment in abduction. Incomplete serial roentgen studies.

Roentgenograms: nine days postpartum—upper end of right femur higher than left. Six weeks postpartum—still high; faint, fan-like callus at upper end of right femur. Ten months postpartum—full restoration to normal. Puppel considered this a case of traumatic dislocation of the hip but later authors (Kleine, Pfeiffer) disagree with him.

Kleine (1933): Version and breech extraction, necessitated by placenta previa in thirty-fourth week of pregnancy. Traction on right foot. On third day, right thigh swollen and painful; right leg shortened and externally rotated. Treated for four weeks with Pappschein's supporting-fixation bandage in abduction and slight internal rotation. Active motion possible after one month and full motion after three months.

Roentgenograms (Fig. 8, *F*): Five days postpartum—upper end of right femur displaced upwards and outwards; slight amount of callus. Nineteen days postpartum—large callus mass around upper end of femur; subperiosteal reaction along proximal third of shaft. One month postpartum—callus increased in size, density and compactness. Three months postpartum—callus absorbed and organized. Six months postpartum—slight elevation, bowing and widening of upper end, indicating traumatic coxa vara deformity; ossific nucleus of head present on affected side. Seven months postpartum—coxa vara; advanced growth on injured side, with larger head nucleus, narrower growth disc and increased width in region of trochanters.

Snedecor et al. (1935): Case I. Version and breech extraction. Forty days after birth, pain and limited motion noticed in thigh. No treatment.

Roentgenograms (Fig. 8, *N*, *O*): Forty days postpartum—callus around upper end of femoral diaphysis; subperiosteal new bone formation along upper fourth of shaft. Four months postpartum—callus absorbed and organized and upper end of femur remodeled; traces of injury still present.

Case II. Version and breech extraction. Soon after birth, left leg swollen, discolored and painful. No treatment. Home follow-up stated that baby recovered completely.

Roentgenograms (Fig. 8, *P*, *Q*): Five days postpartum—no displacement or callus. Twenty-four days postpartum—large callus around upper end of left femur; subperiosteal reaction around proximal fourth of shaft.

Pfeiffer (1936): Case I. Version and breech extraction. Right upper arm fractured at birth. Later parents noticed swelling of left hip. Eighteen days after birth, marked swelling, loss of active motion, painful passive motion. Planned treatment not carried out because baby soon died of pneumonia.

Roentgenogram: About eighteen days postpartum—upward and outward displacement of upper end of femoral shaft; callus mass surrounding it.

Case II. Version and breech extraction. Immediately after birth, right thigh swollen, discolored, crepitant, externally rotated, and immobile. Of sixteenth day postpartum, swelling diminished and plaster cast applied in the Lorenz primary position (for about two weeks). Two months after injury examination showed free motion in affected hip.

Roentgenograms (Fig. 8, *H*, *I*): Three days postpartum—upper end of right femur displaced outwards and upwards; no callus visible. Sixteen days postpartum—beginning callus formation. Four weeks postpartum—still outward displacement; large callus around proximal end of diaphysis; prominent subperiosteal reaction along upper third of shaft; ossific nucleus of femoral head already discernible on injured side. Fourteen months postpartum—ossific head epiphysis larger on injured side than on the other; head epiphysis correctly placed in acetabulum, both sides; no coxa vara deformity.

Case III. Version and breech extraction. Referring physician had diagnosed "dislocation-fracture" and applied cast. First seen by Pfeiffer six weeks postpartum; right thigh then swollen and right leg visibly shortened. Plaster cast with right hip in abduction and flexion (Lorenz primary position) tried but deemed unsatisfactory as promoting development of coxa vara. Then longitudinal tibial traction until displacement corrected, followed by cast with hip in abduction. About four and one-half months after birth, active motion when cast removed. At seven months hip appeared normal but still slight limitation of abduction.

FIG. 8. Diagrams of several cases from the literature.

A, *B*, and *C*, evolution of callus, at first flaky and finally compact and organized.

D and *E*, marked displacement of proximal end of diaphysis.

F, very early appearance of callus (*c*), five days after birth.

G, after Kleine's photograph of baby three days following birth injury.

H and *I*, premature ossification of capital epiphysis on injured side, as early as four weeks after birth.

J, *K* and *L*, characteristic stages of lesion.

M, diagram of Meier's autopsy specimen: *c*, callus; *f.n.+c.*, osseous growth of femoral neck plus callus; *s-p.r.*, subperiosteal new bone formation; *j.c.*, intact joint capsule; *l.t.*, avulsed cartilage of lesser trochanter.

N, *O*, *P* and *Q*, Snedecor's 2 cases, called "traumatic ossifying periostitis" but showing characteristics of other cases of epiphyseal separation.

Roentgenograms: About six weeks postpartum—moderate abduction, proximal end of femur displaced upwards and outwards; small amount of callus. About eight weeks postpartum—displaced higher; in Lorenz position; apparent severe coxa vara; more callus visible. About five and one-half months postpartum—proximal end of femur widened in region of trochanters; ossific head epiphysis already present on affected side and correctly placed in acetabulum. Seven months postpartum—normal position of head and shaft on each side; no apparent coxa vara deformity; head epiphysis larger and cartilaginous growth disc narrower on injured side; still slight thickening of upper end of femur in trochanteric region.

Meier (1939): Case 1. Breech (footling) presentation and extraction. Traction on left foot because umbilical cord caught between legs. Delivery not difficult nor prolonged, however. Four days after birth, swelling and limited motion of left thigh. Nine days after birth, swelling, abduction, external rotation, and slight crepitus on passive motion. Treatment—form of extension with 90° flexion at hip and internal rotation. At four months of age no limitation of motion, at eight months patient stood alone, and at one year patient walked alone, without functional impairment.

Roentgenograms (Fig. 8, *J*, *K*, *L*): Ten days postpartum—upper end of left femur displaced upwards and outwards and externally rotated; on close inspection, faint callus formation and periosteal reaction could be seen. Five weeks postpartum—large callus mass around proximal end of femur; wide zone of subperiosteal reaction along medial and lateral borders of upper three-fourths of shaft. Ten weeks postpartum—callus denser and more compact; subperiosteal new bone formation advanced. One year postpartum—upper end of left femur thickened; head epiphysis larger and growth disc narrower on injured side; increased growth in lesser trochanteric region on injured side and possible ossification of lesser trochanter nucleus.

Case 11. Version and breech extraction. Premature birth, transverse position, prolapsed cord. Weight at birth: 1800 grams. Painful swelling of right hip detected four weeks after delivery; also external rotation and 1 cm. shortening of right leg. No treatment. Infant died at eight weeks of erysipelas. Roentgenogram similar to first case but changes less extensive.

Autopsy findings: Femoral diaphysis externally rotated almost 90° in relation to the neck. Upper femoral epiphysis slipped medially on the top curved surface of diaphysis. Angle between neck and shaft narrowed 4 to 5°. Callus at proximal end of diaphysis. No changes in actual head cartilage. Joint capsule and ligamentum teres intact. No increased joint fluid or old hemorrhage into hip joint.

ANATOMY AND PATHOLOGY

At birth the upper femoral epiphysis is a single large mass of cartilage surmounting the slightly convex upper end of the shaft (Fig. 7). From it are derived the head and both trochanters. The osseous nucleus of the femoral head does not appear till the fifth to seventh month of life (Hodges). The nucleus of the greater trochanter does not begin to ossify until the third or fourth year. In the meantime growth of the femoral neck proceeds steadily, lengthening the distance between head and greater trochanter until in a four year old child, they are completely separated (Fig. 7, *C*, *D*, *E*). At this time there is a definite growth disc of cartilage between each of these bony epiphyses and the neck. The growth of the neck also separates the head from the cartilage of the lesser trochanter. The hip joint capsule is attached to the femoral neck beyond the head; at birth the attachment is proximal to the junction between bony diaphysis and the upper cartilaginous epiphysis. In early years the periosteum over this junction is very tough and strong.[13]

When violence is exerted at birth on the upper end of the femur the cartilaginous mass as a whole is displaced medially and downward off the curved upper surface of the diaphysis. The periosteum is stripped up for a variable distance down along the shaft, sometimes as far as the distal end. There is hemorrhage at the site of epiphyseal separation and beneath the torn-up periosteum. The joint capsule and ligamentum teres remain intact, however, and the cartilaginous femoral head is lodged in the acetabulum. No dislocation of the hip joint is therefore present (Fig. 8, *M*).

Reparative processes begin immediately, marked by the swift production of bony callus. This callus is usually abundant and forms a large, irregular mass around the proximal end of the diaphysis and the base of the displaced epiphysis. There is a larger amount, as a rule, on the upper outer aspect than on the inner. A great deal of the callus (especially in severe cases) lies outside the raised periosteum. Beneath the latter, new bone formation takes place rapidly. The next steps are the absorption and organization of the callus and subperiosteal osseous tissue. The club-like mass gradually disap-

ligament; in another, by combining traction with moderate rotation; and in a third, by using traction plus abduction and rotatory movements. Fixation of the pelvis was a prerequisite. Gurlt's experiments are cited by Ehrenfest and Meier as showing that the epiphyseal separation could be produced only by strong traction together with rotation or abduction.

CLINICAL CHARACTERISTICS

The obstetrician is usually unaware at the moment that the limb has been injured. He can often hear the "crack" when a bony shaft is broken but no audible sound attends the separation of the epiphysis. Pforte, it is true, is said to have heard a "thud" but his case is the exception. The obstetrician in Nathan's case had a "feeling that something was broken" and Harrenstein, in his experiments, "felt something give way."

In almost every instance, however, the injury is not apparent till the delivery is completed. Then it will be noticed that the baby does not move one leg. Swelling of the thigh may or may not be present so soon. If the first is overlooked and the second is slight or absent, the injury may go undetected for a few days but by that time many signs are present, loudly proclaiming it. These are: (1) marked, firm swelling of the affected hip and thigh; (2) slight but noticeable shortening of that leg; (3) no active motion at the hip; (4) pain on attempts at passive motion; and (5) external rotation. Also present sometimes are: (6) bluish-red discoloration of the thigh; (7) crepitus with passive movements; (8) abduction or (9) adduction. The shortening, external rotation and abduction have occasionally been noticed at the time of birth.

The swelling gradually subsides, lasting from two weeks to two months. In its place a "bony thickening" can be felt in the upper thigh, caused by the callus mass around the upper end of the shaft. After about a month partial active motion is usually resumed and after two to five months free motion. The latter, however, may be postponed in the severer cases.

ROENTGENOGRAPHIC CHARACTERISTICS

The first roentgenographic sign is *displacement of the upper margin of the femoral diaphysis*, always outward and usually always upward. This can be ascertained by comparison with the structures of the other side and by noting the position of the proximal end of the shaft in relation to the Y-shaped fissure of the acetabulum. In a few severe cases the end of the shaft is seen above the upper limit of the Y-fissure, lateral to the lower portion of the ilium, but usually it lies below or at the level of the fissure. In most instances the diaphysis is externally rotated; it may be either abducted or adducted.

Faint *callus* shadows can be discovered during the second week, though in one instance (Kleine's case, Fig. 8, *F*) as early as the fifth day. After the first two weeks the callus is readily visible, usually abundant and forming a club-like mass around the proximal end of the diaphysis. At first it is flaky or cloudy but later (fourth week and following) it becomes more compact, more dense and more heavily calcified.

At about the same time as the callus the characteristic sign of *subperiosteal new bone formation*, a result of the avulsion of the periosteum from the cortex, makes its appearance. This extends distally from the upper end of the shaft for variable distances, often beyond the mid-point and occasionally to the lower end. As the callus becomes denser, so does the subperiosteal tissue. The upper limits of this periosteal reaction can often be seen, incorporated in the mass of callus.

During the second and third months the callus and subperiosteal new bone undergo *organization and absorption*. The club-like mass becomes progressively smaller, its edges smoother and its internal structure trabeculated. Reconstruction and remodeling of the femoral neck and upper diaphysis are apparent in the roentgenograms. There may still be outward and sometimes upward displacement of the diaphysis, dependent on the severity of the injury and the subsequent treatment.

Another characteristic sign is the *pre-*

pears and the femoral shaft and neck are reconstructed and remodeled, aided by the osseous growth of the neck proceeding regularly upward from the diaphysis. As a matter of fact, the growth of the head and neck in such an injured femur is accelerated, temporarily at least, and it is common for the nucleus of the head to ossify prematurely. When the bony nucleus of the opposite capital epiphysis appears, it is seen to be obviously smaller than its fellow on the injured side, and the cartilage space or growth disc between nucleus and neck on the sound side is considerably wider.

Part of the above knowledge has been obtained from study of roentgenograms and part from examination of specimens dissected at autopsy. Meier, for instance, described the pathological changes present in an infant dying of erysipelas eight weeks after the birth injury (Fig. 8, *M*). He found the diaphysis of the affected femur externally rotated almost 90° in relation to the neck. The upper cartilaginous epiphysis had slipped medially on the top curved surface of the diaphysis, narrowing the angle between neck and shaft by about 5°. There was bony callus at the proximal end of the shaft. No changes were present in the cartilage of the head itself and the joint capsule and ligamentum teres were intact. There was no evidence of increased joint fluid or of previous hemorrhage into the joint cavity. The cartilaginous lesser trochanter had apparently been avulsed along with the periosteum.

Harrenstein had previously demonstrated the results of this injury produced experimentally in dead newborn infants. The cartilaginous upper epiphysis was separated, all in one piece, at its junction with the diaphysis. The periosteum was separated from the cortex; the joint capsule was intact and not perforated.

CAUSE OF INJURY

All writers are agreed that an unusually forcible manipulation of the infant's leg is the cause of the injury. This is also the case in birth fractures of the femoral shaft or separation of the lower femoral epiphysis. In roughly half the cases of shaft fractures delivery has been by breech extraction, often following version, though some have occurred during cesarean section or "normal" delivery. Among the mechanisms cited by Truesdell[20] are: (1) in the course of podalic version, injury to the femur *in utero* while trying to pull down a leg; (2) torsion of the leg in the vagina (footling); (3) injury while extricating the infant during a difficult cesarean section; (4) abnormal positions and strains on the leg or legs through too vigorous manipulation during difficult extraction of the after-coming head, and (5) even injury after delivery while swinging the child by the leg to promote respiration.

On the other hand, *every case but one* of separation of the upper femoral epiphysis reviewed herein occurred during breech presentation and extraction or version and breech extraction. Although some of the case reports mention no particular difficulty or maneuver, several others note a "difficult" or "prolonged" delivery and "strong" or "powerful" traction on one of the legs. In one, the lower part of the infant's body was manipulated forcibly in trying to deliver the shoulders. In another, a foot was wedged between fetal head and maternal pelvis. The humerus of one baby was also fractured. Unequal traction was used in one instance because the umbilical cord was caught between the legs. In 2 cases there may have been an element of haste—one with placenta previa and one (premature) with a prolapsed cord.

In our case, the lone exception, there was vertex presentation and occipitoposterior position. Difficulty was encountered in delivering the head but none, as far as we can find, in extracting the legs. It is still possible that the legs were forcibly manipulated during attempts to resuscitate the child, if such were necessary.

Harrenstein, in his experiments on dead newborn infants, could not produce epiphyseal separation by traction alone. He was successful in one trial by using strong traction and at the same time forcing the femoral head against the taut iliofemoral

mature ossification of the capital epiphysis on the injured side. This appeared as early as the twenty-eighth day in one case (Pfeiffer, Case II, Fig. 8, *I*, and at three months in another (Truesdell, Fig. 8, *C*). After the opposite nucleus is ossified, it is seen in the roentgenograms to be smaller than the one on the injured side, as late as one to one and one-half years after birth (Fig. 10). The cartilaginous growth disc likewise is narrower on the affected side, more evidence of accelerated growth in that region. When the bony nucleus of the injured femur becomes visible it is seen to be correctly lodged in the acetabulum.

At later examinations there is usually only widening of the proximal end of the diaphysis in the trochanteric region; traces of the old callus and the remodeling process may still be present. There is often a coxa vara deformity, with narrowing of the neck-shaft angle; the upper third or fourth of the shaft may be bowed in a varus manner. In some instances—the least severe cases—the bones appear normal.

PROGNOSIS AND TREATMENT

Lesser degrees of injury are usually followed by good end-results. This may be true without any treatment (Snedecor's first case) or with only moderate treatment (Puppel, Harrenstein, Case I; Pfeiffer, Case II). Severer injury often results in a coxa vara deformity, especially if no treatment has been given (Harrenstein, Case II; Meier, Case II). Moderate treatment in Kleine's case did not forestall the development of a considerable coxa vara; rather extensive measures in 2 other cases (Pfeiffer, Case III; Meier, Case I) did prevent this deformity.

Pfeiffer discusses the prognosis at length. He separates the cases into two groups: (1) less severe injury, with good prognosis; and (2) severe injury, with poorer prognosis. In the *first* group (Fig. 9, *A*, *B*) roentgenograms show only a moderate outward and slight upward displacement of the proximal end of the diaphysis. Presumably the perichondro-periosteal membrane has not been ruptured but only lifted up (a point put forward by Harrenstein), thus preventing any great displacement of the fragments. These are the cases with good end-results despite no treatment or only gentle supportive measures.

In the *second* group (Fig. 9, *C*, *D*) roentgenograms show a greater deviation outward and a marked displacement upward.

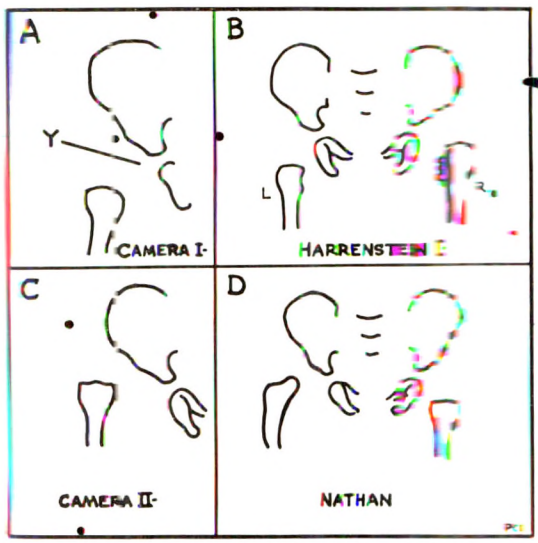


FIG. 9. Diagrams after Pfeiffer.

A and *B*, mild injury and good prognosis. The proximal end of the femoral diaphysis lies below the Y-shaped fissure of the acetabulum (*Y*).

C and *D*, severe injury and poorer prognosis. The proximal end of the diaphysis is above or on a level with the Y-fissure.

The upper end of the diaphysis is found above the Y-fissure and sometimes even above the upper rim of the acetabulum. The soft tissue injury here must have been very great, associated with tearing of the periosteum and severe damage to the surrounding muscles. In these cases coxa vara is almost certain to develop unless prompt and thorough orthopedic treatment is instituted. Pfeiffer cites his own third case, Camera's second, and Kleine's single case as illustrations of the second group (Fig. 10).

Our own case falls into this second class with respect to the degree of displacement and apparent severity of injury; we cannot tell yet whether coxa vara deformities are

developing, though they are likely to be. Treatment of the hips has just begun (five months after birth).

The orthopedic procedures described in the literature have not been uniform (see Table 1 and case reports).

DISCUSSION

Perhaps the diagnosis of traumatic separation of the upper femoral epiphysis will be made more often in the future. This will come about if the obstetrician examines the infant more closely after podalic version and breech extraction and if serial roentgenograms are taken in suspected

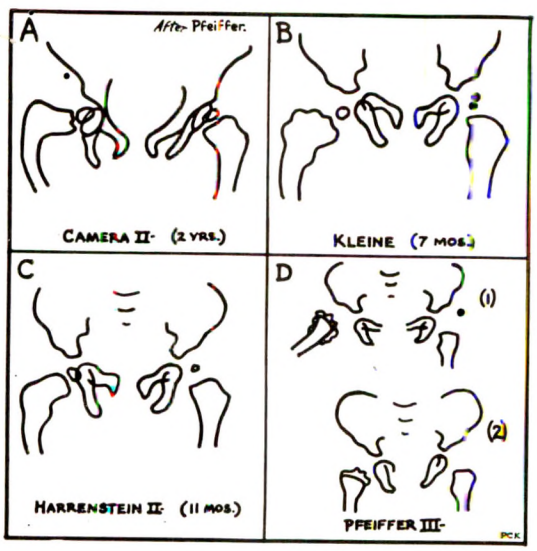


FIG. 10. Diagrams illustrating end-results.

A, *B*, and *C*, varying degrees of coxa vara. Note the larger capital epiphysis and narrower growth disc on the affected side.

D, good result following orthopedic treatment. (1) Unsatisfactory position after trial in Lorenz primary position. (2) Good position after extension and tibial traction.

cases for at least three weeks after birth. In the first week the roentgenologist should detect the displacement of the upper end of the diaphysis. The next roentgenograms, a week or two later, will show the early callus and confirm his tentative diagnosis.

It is just as important to follow the later course of the lesion by serial roentgenograms. As the callus increases and then is absorbed and organized, the reconstruction of the femoral neck and upper shaft can be observed and appraised. The development of a coxa vara deformity can be foreseen and appropriate measures taken to avert it. Further, the effects of treatment can be noted and evaluated.

Serial roentgenograms will also rule out the remote possibility of a traumatic dislocation of the hip occurring at birth. Such a lesion has long been the subject of controversy, most authors denying its existence. Nathan and Puppel, mentioned above, considered their cases examples of traumatic dislocation. One of Kleine's cogent reasons why Nathan's case was really an epiphyseal separation is that Nathan did not see the patient until three and one-half months after birth, when the callus was all gone and the roentgenogram showed only upward displacement and bowing of the proximal end of the diaphysis. Likewise, Kleine's objection to Puppel's diagnosis is that there were no roentgenograms between the ninth and forty-second days. On the former date the right femur was displaced upward—the only sign; on the latter a faint fan-like callus was present (though apparently missed by Puppel). Roentgenograms in the interval would probably have shown the characteristic development of the callus, a phenomenon not found in dislocation of the hip.

Observation of later roentgenograms provides another argument against dislocation of the hip. When the nucleus of the femoral head ossifies it is seen to be in its normal position in the acetabulum. Moreover, as Kleine points out, osseous growth of the affected capital epiphysis and femoral neck are accelerated in these cases of epiphyseal separation; on the contrary, hypoplasia is the rule in dislocation of the hip joint.

SUMMARY

(1) Traumatic separation of the upper femoral epiphysis is a rare birth injury. Twenty cases from the literature (mostly foreign) have been reviewed and our own case report added.

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AN OSCILLATING PLEURAL HERNIA*

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A PLEURAL hernia shifting through the mediastinum is not a strange or unknown entity to those specialists doing many pneumothoraces, or a large amount of roentgenography. A reversible pleural hernia, which swings pendulum like across the median line, from right to left, is however an exceedingly interesting phenomenon.

A thorough search through modern literature reveals only 11 cases of transmediastinal hernia reported. To this list we wish to add another which is believed to be the twelfth case of oscillating pleural hernia recorded. The first mediastinal hernia was reported by Cantieri[2] in 1925. Subsequently, single cases have been reported by Bordet and Parodi,[1] Locatelli,[6] Raimondi and Sangiovanni,[7] Timpano,[9] Rey, Rey and Pucci,[8] Dufourt, Muller and Jandaud,[3] Lev-Valensi and Charles;[5] Finkelstein[4] reported 3 cases.

There are two sites where these hernias are likely to occur. One is located in the superior mediastinum and anteriorly. In this area the air pressure pushes the pleura to the opposite side directly behind the sternum. The second location of transmediastinal hernias is usually in the posterior mediastinum, inferiorly. Here the hernia occurs between the esophagus and the arch of the aorta.

REPORT OF CASE

E. B., white, male, aged thirty-four.

Family History. Father and mother dead; ages and causes unknown. Two brothers and one sister living and well. One brother died in infancy. Wife living and well. No cancer, tuberculosis, neuropsychiatric or cardiac disease known in any members of family.

Personal History. Measles, pertussis and smallpox during childhood. No illness in early adult life. Denies syphilis. Gonorrhea in 1919. No recurrence or residuals. Smokes cigarettes moderately. Denies the use of alcohol or drugs. Has had no operation. Fractured right elbow at age of five or six.

Military History. Enlisted in Army June 2, 1918; discharged May 13, 1919. Not overseas. Hospitalized two weeks early in 1919, with tonsillitis, and cough. No tonsillectomy.

Present Illness. Patient states that in the latter part of 1920 he began to have a cough, soreness throughout the chest; fever for a few days at a time, occurring about twice a year, generally in the spring or fall. Was hospitalized two or three weeks in 1923; two weeks in 1929, and from September 13, 1933, until November 15, 1934. Was sick four months at home in 1925. Began feeling tired all the time. Soreness in the chest; night sweats; loss of weight—lost 21 lbs. in the past six or seven months; coughs some all the day but more at night. Expectoration is moderate. No frank hemorrhage. Sputum contains small particles of blood occasionally. Has had sore throat at times—none in the past two or three years.

Worked in the coal mines in 1920 and continued to 1929. Worked as an attendant in a hospital from 1929 to September, 1933. He lost time for two or three weeks; once for five months in 1920 because of illness.

Sputum positive for tubercle bacilli in 1925.

Chief Complaints. Fatigability, cough and expectoration, dyspnea, loss of weight, anorexia, vomiting, night sweats.

* Published with the permission of the Medical Director of the Veterans Administration, who assumes no responsibility for the opinions expressed or the conclusions drawn by the authors.

(2) Every case except one has occurred during podalic version and breech extraction or breech presentation and extraction. Our case is exceptional in that the position was occipitoposterior, delivery by low forceps, and that no known violence was exerted on the legs. It is also the only case in which the lesion is bilateral.

(3) The upper femoral epiphysis at birth is a single cartilaginous mass, including head, neck and both trochanters. It is displaced medially and downward at its junction with the diaphysis. The periosteum is usually stripped up and sometimes ruptured. The joint capsule and ligamentum teres remain intact.

(4) Rotatory movements combined with traction on the leg are necessary to produce the injury.

(5) The clinical signs are swelling, slight shortening, limitation of active motion, painful passive motion, and external rotation; also sometimes discoloration, crepitus, and abduction or adduction.

(6) The roentgenographic signs are displacement of the proximal end of the diaphysis (upward and outward); rapid, profuse callus formation and prominent subperiosteal reaction around the upper end of the shaft; absorption of callus and reconstruction of the femoral neck; and premature ossification of the capital epiphysis and accelerated growth of the femoral neck on the injured side.

(7) The prognosis is good in cases of milder injury but rather poor when the initial trauma and displacement have been severe. Coxa vara is a frequent end-result in the latter group of cases.

(8) The value of serial roentgenograms is emphasized in the diagnosis and following of these cases.

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Physical Examination.

General appearance: Inspection reveals a fairly well nourished and developed white male, who looks chronically ill. Height 68¼ inches; weight 133½ lb.; average 155–160 lb.

Scalp: Hair normal; no baldness.

Skull: No elevation, or depressions.

Eyes: Right, vision—20/50; corrective lenses—20/20; pupil reacts to light and accommodation. Left, vision—20/50; corrective lenses 20/20; pupil reacts to light and accommodation.

Ears: Right, conversation voice—2 feet; whispered voice—0; drum membrane—retracted, adherent. Left conversation voice—35 ft.; whispered voice—35 feet; Weber test—equal; Rinne, right—negative for all forks, no tones heard, left positive, all tones heard.

Nose: Negative. Breathing space ample.

Face: A few small scars and bluish pigmentation on face, due to coal mine injury five years ago.

Mouth: Mucosa normal.

Teeth: All uppers extracted and most of lowers extracted.

Tongue: Normal.

Tonsils: Small.

Neck: Thyroid not enlarged.

Chest: Medium length, width and thickness; there is slight tenderness on pressure, right base, posteriorly. Mobility fair and equal.

Palpation: Fremitus: right lung—slight increase throughout; left lung—not increased.

Percussion: right lung—impaired resonance above 3rd rib and 7th dorsal spine. Left lung—impaired resonance above 2nd rib and 5th dorsal spine.

Auscultation: Right and left lungs—whispered voice sounds not increased. Breath sounds vesicular. No râles heard.

Gastrointestinal tract: Patient gives a history of constipation extending over a period of several years past. States that the stools are frequently hard and frequently one stool in several days. States that different kinds of remedies have been used regularly about once or twice a week for the relief of constipation over a period of the past three years. No history of vomiting. No hematemesis. No jaundice. Jaundice is not present at this time. States that there is an occasional discomfort in the abdomen, and that this discomfort is general. This discomfort does not bear any relation to meals, according to patient's statement.

Wassermann and Kahn reactions, negative. Erythrocyte count, 4,600,000; leukocyte count, 13,800, with 80 per cent polymorphonuclears; hemoglobin 85 per cent. Urinalysis, negative.

Genitourinary report: The kidneys and bladder region are negative. Genitals, negative. There is no adenopathy. The urine is clear in both portions, and essentially negative for cellular elements. The prostate and vesicles are normal. The prostatic and expressed fluid is negative.

The patient was studied by the surgical collapse board. The right artificial pneumothorax was discontinued, as it was thought the lung had been collapsed an adequate length of time. A pneumothorax was started in the left because of more recent lesion. It was discontinued because of repeated spontaneous pneumothoraces.

Patient has been on bed rest, as he is of the nervous type with multiple complaints, although he is not difficult to handle. The left lung has been completely re-expanded. The heart is retracted to the right, but the right lung is not entirely re-expanded, as shown on the roentgenograms. Pain in the right chest, apparently due to high negative pressure, is relieved by instillation of small amount of air. On examination, the left side is essentially clear. Last sputum examination was positive. Gaffky III.

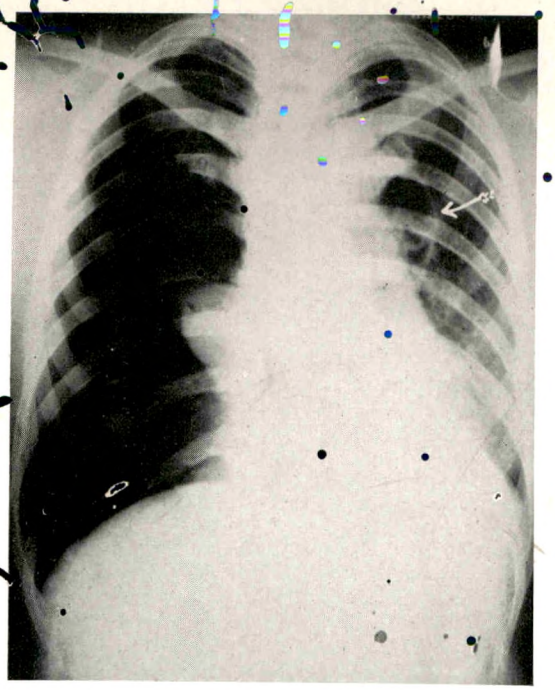
Roentgen Examination.

Gastrointestinal tract: Roentgenoscopy—Opaque meal passed the esophagus without hesitation. Stomach filled normally. Both curvatures are regular in outline and freely movable, exhibiting no filling defects, or deformities. The duodenal bulb filled, showing no evidence of abnormality, spilled over to the right and formed a normal first and second portion of the duodenum.

The roentgenographic examination of the gastrointestinal tract confirms roentgenoscopic findings, showing on the immediate films a well filled stomach and duodenal bulb. The colon is normal. The gallbladder is well visualized, displaying no alteration in appearance to suggest cholecystic disease.

Roentgen diagnosis: Gastrointestinal tract normal; gallbladder normal.

Chest: The roentgenographic examination of the chest at 2 meter distance shows the bony framework to be barrel shaped and symmetrical, exhibiting no kyphosis, cervical, or forked regions.



Right Left

FIG. 1

Domes of both diaphragms are smooth. Costophrenic angles are deep, acute and clear, displaying no evidence of fluid, adhesions, or thickened pleura.

The heart image is of normal size and shape and position for the type chest possessing it. Borders are regular; measurements are not accentuated, and there is no roentgen evidence of organic heart disease.

The lobes of the right chest are totally compressed by a pneumothorax. The right dome of the diaphragm is depressed. The heart, thoracic and mediastinal contents are also displaced to the left. A semicircular shadow line is seen extending from the region of the first anterior rib outward in a graceful curve and ending in the region of the upper border of the third anterior rib, attributed to a pleural hernia (Fig. 1).

The lobes of the left chest show slight accentuation of the bronchial tree markings, with interstitial fibrosis in the right upper. The vertebral first and second interspace trunks are prominent, fuzzy, beaded and interweaving, and apparently reaching the periphery, attributed to an inflammatory process. This snow storm effect mimics the findings of tuberculosis.

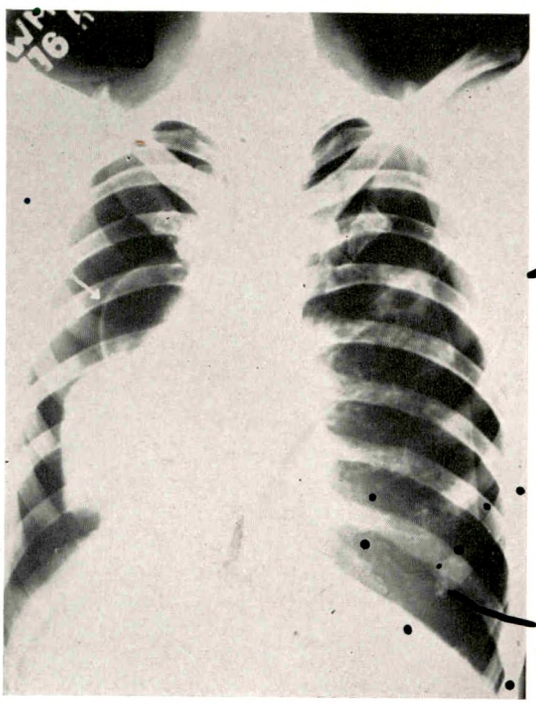
The examination of the chest roentgenogram taken two years later shows a total collapse of the lobes of the right chest and also a snow storm density infiltrating the left upper lobe and apex. A pneumothorax in seen in left chest extending from the apex to the base and occupying the peripheral lung zone, producing approximately 25 per cent collapse, lobes of left chest.

A semicircular shadow line is seen in the right upper chest extending from the region of the first anterior rib outward in a graceful bulging shadow line and ending in the region of the upper border of the third anterior rib, attributed to a pleural hernia (Fig. 2).

The pleural hernia, seen on previous roentgenogram, in the left root lung zone has now reversed itself to the root lung zone of the right hemithorax.

Evaluating the roentgenographic findings displayed in this interesting phenomenon, wherein the pleural hernia of the left chest is observed, and demonstrated to shift to the right hemithorax, this is believed to be caused by the incarcerated air in the right pneumothorax cavity having been absorbed, thereby creating a large vacuum or an extensive negative pressure area.

The small artificial pneumothorax occupying the peripheral lung zone of the left chest was



Right Left

FIG. 2

therefore sufficient pressure to displace the pleural hernia to the opposite side, thereby causing this exceedingly interesting phenomenon.

The authors are indebted to Parvin M. Eaton for his aid in preparing the illustrations accompanying this article.

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A SIMPLIFIED METHOD OF BRONCHOGRAPHY

By MAJOR PERCIVAL A. ROBIN

[illegible]

SEVERAL standard procedures for the instillation of iodized oil into the bronchial tree are in general use but all methods have certain drawbacks and do not adapt themselves well to the needs of a military roentgenology department. One of the early methods advocated was to introduce the tip of the cricothyroid membrane, having previously anesthetized the skin locally with 5 per cent novocaine intratracheally. The preliminary preparation for this method is time-consuming and requires careful aseptic technique. In addition, there is a constant danger that the oil will escape into the fascial planes of the neck, and even gravitate into the retrosternal tissues where it remains for years. Dysphonia, edema of the glottis, dysphagia and subcutaneous emphysema are constant hazards with this technique.

In some institutions it is the practice to allow the otolaryngologist to introduce the iodized oil, either via the bronchoscope or directly into the larynx, before sending the patient to the department. This method seems to be inadequate, for as a rule the roentgenograms disclose a very limited visualization of one segment of the bronchial tree, and in most cases sufficient time has elapsed before the roentgenograms are taken to permit the oil to find its way into the alveoli. When the latter happens, no matter what method is used, accurate interpretation is out of the question. Clinically unsuspected lesions, therefore, are overlooked because of the segmental visualization.

The most universally employed routine is the transglottic method, using a curved catheter tip. The preliminary preparation requires complete anesthesia of the pharynx and larynx with 5 per cent cocaine and adrenalin. During the introduction of the oil it is necessary for the patient to grasp the tongue firmly with one hand, enhancing the grip with gauze. Theoretically, this opens the glottis automatically and allows the oil to collect in the larynx where it is aspirated [illegible] and flows into the bronchial tree by gravity.

The several disadvantages of this method can be summarized as follows: There is a certain element of danger in the employment of cocaine but, if inadequate amounts are used, the anesthesia is not complete enough to prevent the occurrence of the gagging reflex. The reflex is very easily touched off in the darkened roentgenoscopic room where the operator, having his attention directed to the flow of the oil into the proper bronchi, is apt to allow the cannula to deviate to the sensitive pharynx. Furthermore, to obtain a complete visualization of the bronchial tree it is necessary to tilt the patient in several positions and in so doing the situation becomes progressively more awkward, for during the entire operation it is essential that the tongue remain immobilized and that the curved cannula, through which the oil is being injected, remain in place.

The simple method proposed here was evolved by necessity over a period of months. It requires no preliminary preparation of the patient and no elaborate equipment and is therefore particularly well adapted to Army use. The only medication is ½ to ¾ gr. of codeine administered about an hour before the patient arrives at the roentgen department, to diminish the cough reflex. After the patient has entered the roentgenoscopic room, a preliminary explanation is given him, describing in detail and simple what is to be done and what is expected of him in the way of cooperation. Two admonitions are stressed repeatedly: that he must not cough during the entire procedure and that he must not swallow, no matter how strong the desire becomes. If the impulse either to cough or to swallow

the diagnosis has been established in such cases, however, an attempt at further filling is unnecessary, unless precise knowledge of the extent of the lesion for surgical evaluation is required.

CONCLUSIONS

A simple, effective, rapid method is described to accomplish bronchography. It is completely adequate and does not require either an elaborate set-up, specialized skill, or protracted preparation.

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THE USE OF A MODIFIED OPAQUE BARIUM SULFATE MIXTURE IN ROENTGENOGRAPHY OF THE COLON*

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THE purpose of this paper is to describe the use of a modified opaque barium sulfate mixture in roentgenography of the colon. This method permits simultaneous demonstration of (1) the luminal contour as available from the ordinary, densely opaque barium sulfate enema, and (2) a view of the normally distended colonic lumen and its contents, if any. In the ordinary method a view of the lumen is not possible, due to its opacification. After evacuation of the modified opaque mixture it is possible to obtain a view of the rugae such as seen after the evacuation in the ordinary barium enema method.

Following success with the use of a modified opaque meal in roentgenography of the gastrointestinal tract,[1] attention was directed to the colon where, after experimentation with various mixtures, it was found that a mixture containing 75 grams of barium sulfate and 5 grams of gum acacia to 2 quarts of water was the most suitable. In this manner sufficient contrast is maintained and, at the same time, the colon assumes a "pearly" or "porcelain" semitransparency which is enough to visualize any abnormal or normal luminal contents. Ordinarily, it would require the instillation of gas or air, with or without a small amount of barium sulfate, to visualize the colon in this state of normal distention.

The method is especially valuable for the study of the colon in the regions of the flexures and sigmoid and in those cases with marked colonic redundancy. One is thus able to resolve the component parts of various loops which are ordinarily clumped and densely opacified. This eliminates any additional oblique views and minimizes the number of lesions in the flexures and sigmoid which may occasionally be overlooked due to superimposition of a normally opacified portion of the colon. Polyps low in the colon are more easily seen, and by their persistence in size, shape and position are differentiated from fecal masses. In the presence of a sigmoid mass it may occasionally be difficult to differentiate between cancer or an inflammatory mass associated with diverticula, or to determine the presence of both conditions. With the modified mixture the diverticula will be more likely to fill.

Roentgenoscopy is more difficult than under ordinary circumstances. This, however, is somewhat balanced by utilizing the full accommodation of the roentgenoscopist before beginning the examination and by the actual roentgenograms. Besides, experience has shown that this thin mixture passes in much more rapidly, so that the total roentgenoscopy time for the examination is reduced. Thus, it may be permissible to increase the milliamperage to a slight extent.

Another finding has been the visualization of numerous fecal masses in cases which would otherwise appear well prepared with the routine method. This proves that very few individuals, even after thorough preparation, are completely evacuated, and that in the ordinary method a coating of barium sulfate sufficiently opacifies and blends the fecal masses with the barium sulfate in the lumen.

In cases of obstruction a small amount of this modified mixture is more likely to get by the narrowed portion to outline the lesion than is the ordinary thicker mixture, for too often the thick barium sulfate mixture halts at the most distal point of ob-

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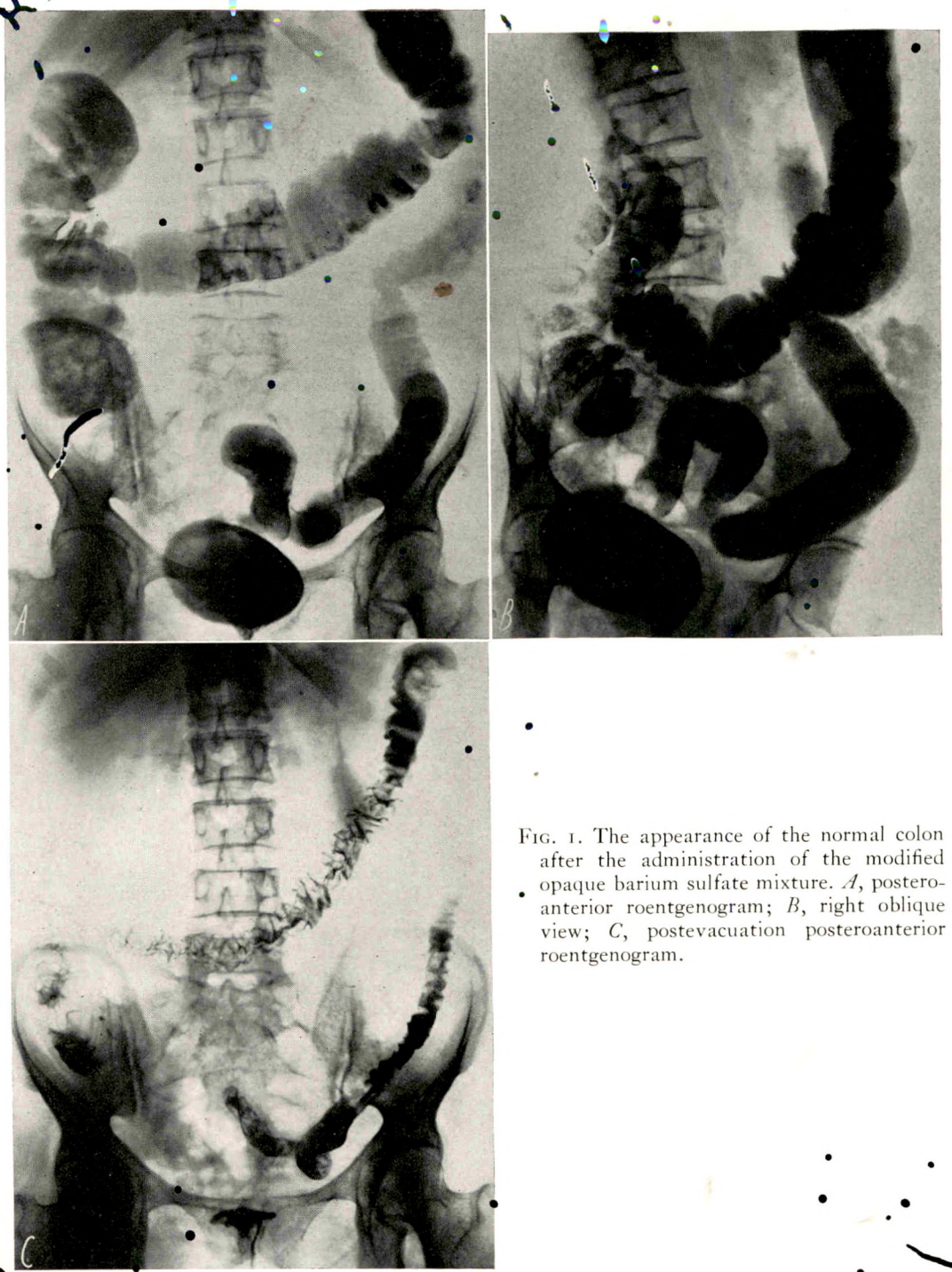


FIG. 1. The appearance of the normal colon after the administration of the modified opaque barium sulfate mixture. *A*, posteroanterior roentgenogram; *B*, right oblique view; *C*, postevacuation posteroanterior roentgenogram.

struction and one can only state that there is an obstruction, and nothing else. Further, it is our distinct impression that patients seem to hold more of this thin mixture before complaining of discomfort.

Correlation between roentgen and pathological studies has usually shown that the lesion pathologically is much larger than has actually been seen on the roentgenogram, despite the distortion factor which

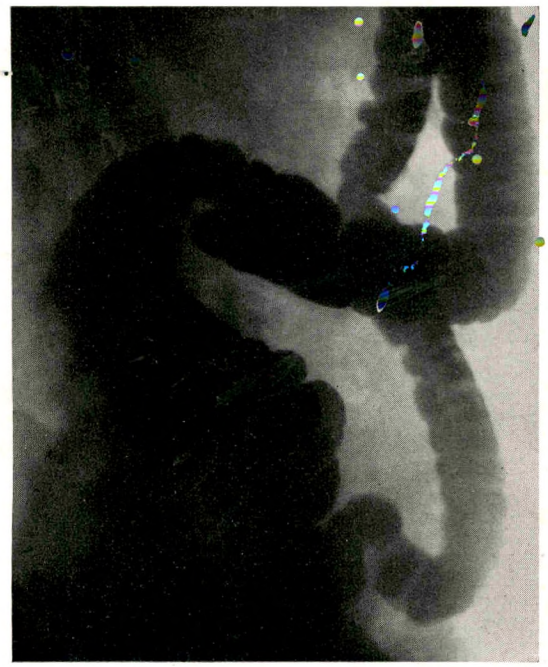


FIG. 2. Right oblique view of barium enema with ordinary barium mixture, showing a mass near the hepatic flexure.

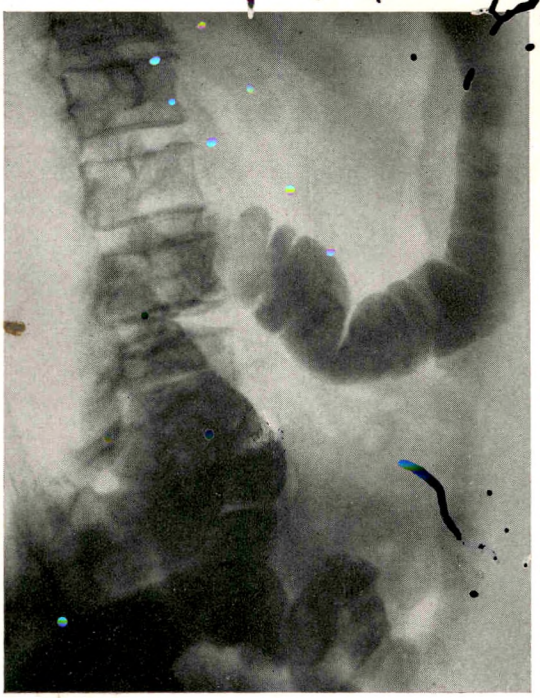


FIG. 3. Same case as Figure 2; roentgenogram made the following day, using the modified opaque barium sulfate mixture. Note the difference in the size of the mass.

actually tends to increase the size of the lesion with the technique ordinarily used for barium enemas. The modified method has resulted in demonstrating a larger lesion than the thicker mixture in the same case during the same day or within twenty-four to forty-eight hours. This is due to the fact that the thick barium sulfate opacifies and thus obscures the edges of the lesion by passing either superiorly, inferiorly, anteriorly or posteriorly to it. This does not obtain in the modified mixture and thus the results more nearly approximate the actual pathological picture.

Occasionally upon the completion of a barium enema with the ordinary method the examiner feels perplexed, and upon re-examination finds no additional information to enable him to reach a conclusion. In such a case it might be advisable to make the enema examination again, with the modified mixture. This different approach may be productive of sufficient information to enable the examiner to arrive at a conclusion.

CONCLUSION

The use of a modified opaque barium sulfate mixture in roentgenography of the colon has been described.

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THE TREATMENT OF ACCESSIBLE MALIGNANT TUMORS WITH SHORT DISTANCE LOW VOLTAGE ROENTGEN RAYS*

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AN advance was made in the method of treatment of accessible malignant tumors by the introduction of modern apparatus for short distance low voltage roentgen therapy. This advance originated with the work of Schaefer, Witte, Chaoul and Adam. Working at first with cathode rays and starting with the assumption that

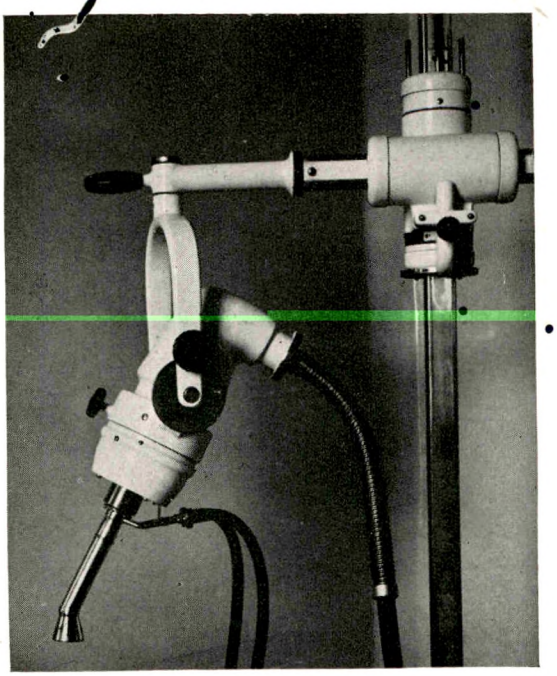


FIG. 1

the success previously obtained with radium was not due to the quality of the radiation but to the distribution of the energy absorption in the tissues, they evolved a method of roentgen treatment which gives a similar distribution. This was achieved by the use of a short focal-skin distance and a low voltage, the object being to confine the radiation effect to a small volume of tissue.

Apparatus. The apparatus (Fig. 1) was made by Messrs. Siemens and was originally designed by Schaefer and Witte for intracavitary roentgen therapy. Its use in the treatment of skin and other accessible tumors was developed by Chaoul and Adam. This tube (Fig. 2) is constructed so that the stream of cathode rays passes down an earthed metal cylinder to strike the target situated at the far end. The roentgen rays used in treatment pass through the anode which is water cooled. In this way the source of the roentgen rays can be brought to within a few millimeters of the lesion to be treated.

More recently Messrs. Philips have constructed an apparatus for short distance roentgen treatment (Fig. 3). The Philips

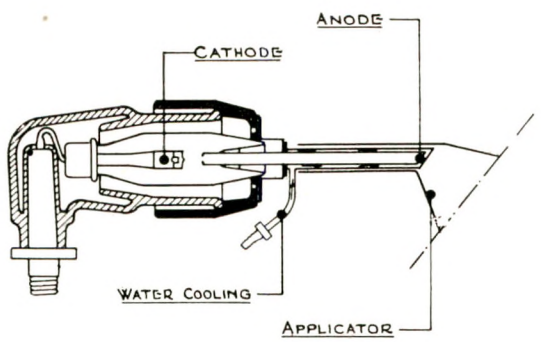


FIG. 2

roentgen tube (Fig. 4) is designed so as to avoid a transmission target and thus reduce the inherent filtration. In this tube the cathode is earthed and the stream of cathode rays passes in the opposite direction to the roentgen rays utilized. The anode is separated from the lesion by a glass window and a "philite" cap and is cooled by radiation to the walls of the tube

* Prepared for presentation with a color film at the International Cancer Congress, Atlantic City, N. J., September, 1939.

which are themselves air-cooled. The inherent filtration of the Siemens tube is equivalent to 6.3 mm. Al and of the Philips tube to 0.2 mm. Al.

Advantages. The main advantages that short distance low voltage roentgen treatment has over the use of radium are the ease of application, the fact that the radiation may be limited to the tumor and its immediate vicinity (the rest of the patient is not irradiated at all), the high intensity of the radiation, the low initial cost of the apparatus, and the freedom from danger to the personnel of the department. The high intensity is of particular importance for it allows large numbers of patients to be treated in a short time by limiting individual treatment times to a matter of a few minutes. Short treatment times also mean a minimum of disturbance with the patients' usual occupations and save the discomfort accompanying prolonged applications to certain parts of the body.

Limitations. This method of treatment is strictly limited in its application to lesions that are directly accessible either

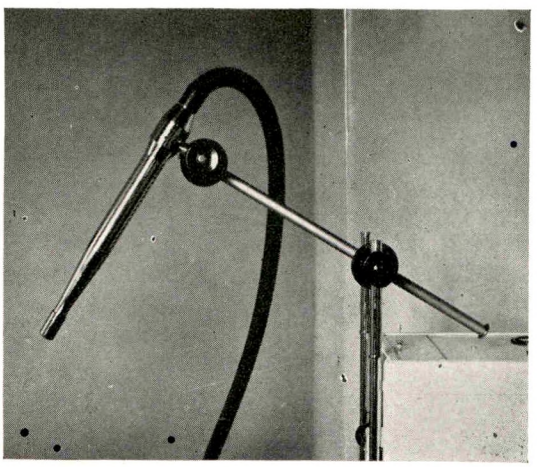


FIG. 3

by virtue of their situation or by means of previous surgical exposure, and are of limited depth. These limitations result from the distribution of the radiation and are an integral part of the design of the treatment.

Method of "Caustic" Roentgen Therapy. The majority of tumors that can be brought into direct contact with an applicator involve the skin or mucous membranes so that consideration of skin tolerance, that important limiting factor in so many roentgen treatments, does not arise. Overdosage in short distance therapy is not often followed by such serious consequences as it is in high voltage treatment for it is con-

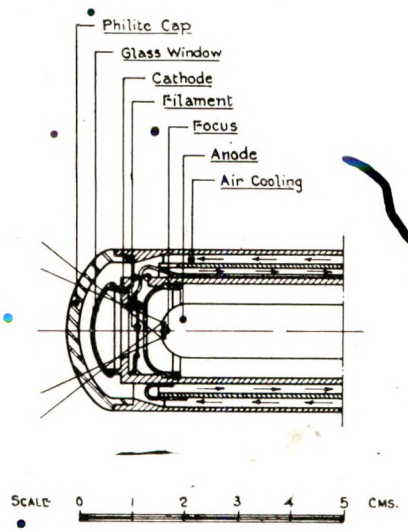


FIG. 4

fined to a small volume of tissue most or all of which is tumor tissue. It was considerations such as these that led Van der Plaats to modify the method of treatment evolved by Chaoul and Adam. By using the Philips tube with its low filtration he was able to confine the energy absorption to a still smaller volume of tissue and so reduce still further the risks of overdosage. The object of his treatment is to give a sufficient dose to the base of the tumor without any consideration of overdosage to the surface but with as little damage as possible to the underlying healthy tissues. For this purpose he uses those factors which provide the steepest decrease of dose with depth obtainable but which allow the application of a sufficient dose at the point intended in a reasonable time. With this method the dose at a depth of 0.5 cm. may be only 40 per cent of the surface dose and the dose at 1.0 cm. depth less than 20 per cent. The

object of treatment is to "burn away" the tumor and yet preserve the surrounding normal tissues and is emphasized by the fact that it is called the "caustic" method of roentgen therapy.

Time Spacing of the Radiation. Following the common practice of high voltage roentgen therapy, Chaoul and Adam adopted fractional irradiation in their treatment. Van der Plaats and others who employ the "caustic" method of roentgen therapy have abandoned fractional irradiation in favor of single massive doses on the grounds that they had difficulty in persuading their patients to attend regularly for treatment to lesions that they considered to be trivial, and further that there was in fact nothing to be gained by fractionation of the dose in this type of treatment. For some very small tumors single doses are effective, but neither the symptom-free rate nor the cosmetic results are as good as those obtained with divided doses and further consideration is necessary before we abandon the established principle of fractionation.

Accessible malignant tumors may be treated either by surgical excision or by destruction *in situ* by irradiation. Success with surgery is dependent upon a sufficiently wide margin of surrounding tissue being removed with the tumor to avoid the possibility of leaving any malignant cells behind. Treatment by radiation relies for its effect upon the malignant cells being more susceptible or less adaptable to this form of injury than normal cells. Excision produces deformity for, by its very purpose, it cannot allow the cosmetic result to be more than a secondary consideration. Deformity following irradiation should be minimal for it is almost entirely due to the previous destruction of the normal tissues by the tumor. Fractionated irradiation is employed to take advantage of the selective action on dividing cells and increase the advantages of irradiation methods over surgical, by minimizing the risk of dissemination and the amount of deformity. As our knowledge of the correct spacing of radiation treatments improves, these advantages will gain even greater importance. Treatment by a single massive dose of radiation sacrifices some of these advantages, it aims at dealing a "vital blow" to the tumor and so falls into an intermediate position between fractionated irradiation and excision. Roentgen therapy aims at destroying the tumor's capacity for proliferation; the dose required is the minimum dose that will prove effective fractionated according to the cell composition and response to treatment of each individual tumor.

Objections to the "Caustic" Method of Roentgen Therapy. The accuracy with which it is necessary to estimate tumor size depends, in practice, on the rate of change of dose with depth in the tissues. It follows that the more we reduce the penetration of our beam the more accurate must our estimation of tumor size become. From tables published by Van der Plaats it may be seen that, under one set of conditions, to deliver a dose of 3,000 r at a depth of 0.5 cm. necessitates a surface dose of 7,500 r, and to deliver the same dose at a depth of 1.0 cm. requires a surface dose of 15,400 r. A difference in depth of 0.5 cm. may therefore mean a change in surface dose of nearly 8,000 r. The intensity of the radiation under these conditions is more than 7,000 r per minute. With such a high intensity and with so rapid a change of dose with depth it is not easy to obtain accurate dosage for the following reasons: (*a*) the very high dosage rate makes the attainment of "saturation" in the ionization chamber difficult, (*b*) the short treatment times necessitate very accurate timing and (*c*) owing to the rapid change of dose with position in the irradiated mass, ionization chambers of normal dimensions will not accurately follow the dosage gradients.

The "caustic" method of roentgen therapy aims at delivering a prescribed dose to the base of the tumor. The base of a tumor is formed by malignant cells spreading out and invading the normal tissues. It is a region in which the percentage of malignant to normal cells is usually decreasing with depth, though the behavior

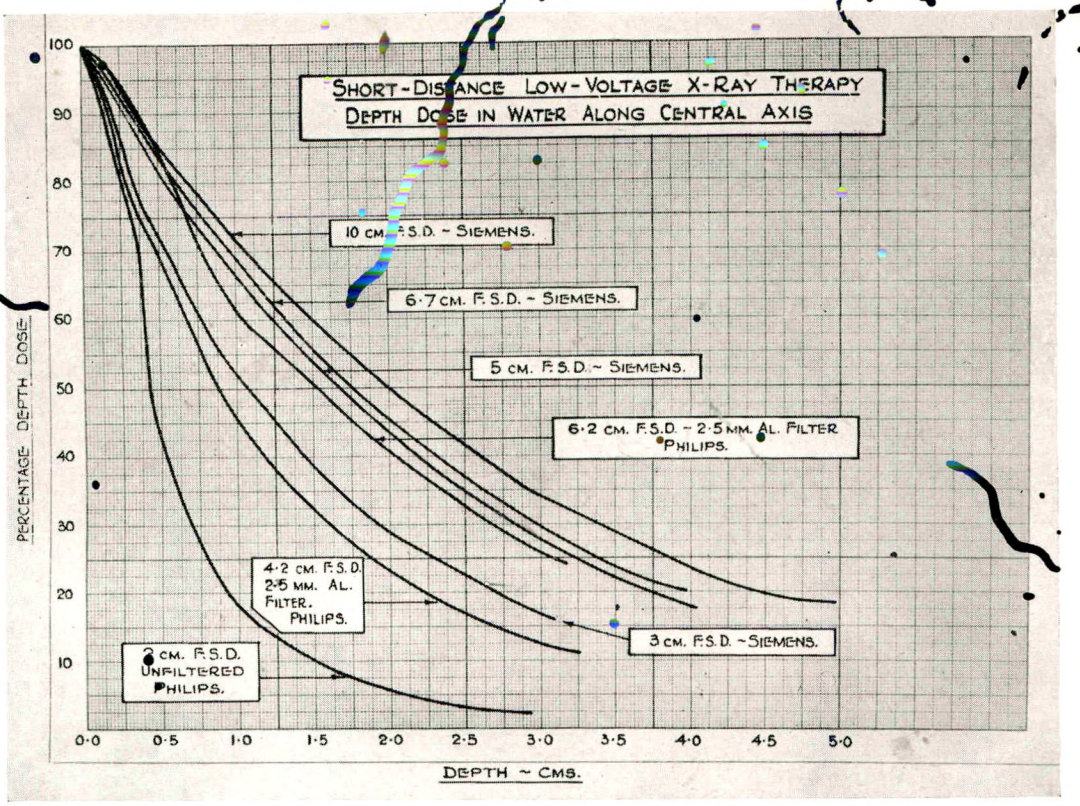


FIG. 5

of the tumor in this region is variable and can only be seen on microscopical examination. For each case treated we must presuppose a change from tumor to normal tissues in the depth commensurate with the rapidity of the change of dose with depth employed. The "caustic" method of roentgen therapy reduces the limits of safety in delivering an adequate dose to the furthermost extension of the tumor. The protagonists of this method have laid great stress on the preservation of the normal tissues from which regeneration must start but healing has been shown to be extremely satisfactory with the methods advocated by Chaoul and Adam so that insistence on still more rapid change of dose with depth in most cases of accessible cancer is unnecessary.

The "caustic" method allows no more than a mere approximation to the dose delivered to the tumor bed. As an irradiation method it sacrifices some of its margin of safety as well as some of its cosmetic advantages to speed and simplicity. It is doubtful whether this is justifiable in the treatment of any but the smallest tumors or except under special circumstances, to be referred to later where speed is of particular importance.

Royal Cancer Hospital Methods. At the

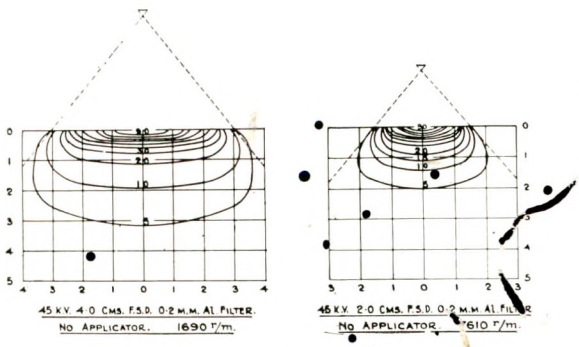


FIG. 6

Royal Cancer Hospital we make use of both the Siemens and the Philips apparatus which together provide a whole series of distributions of the radiation in the short distance low voltage therapy range. These vary from that of the Siemens with a 10 cm. focal-skin distance to the Philips with a 20 cm. focal-skin distance (Fig. 5). Treatment is designed on the assumption that the smaller the tumor the less the need for penetration, and the larger the tumor the more the need both for penetration and for fractionation. The most suitable distribution is employed for each particular case.

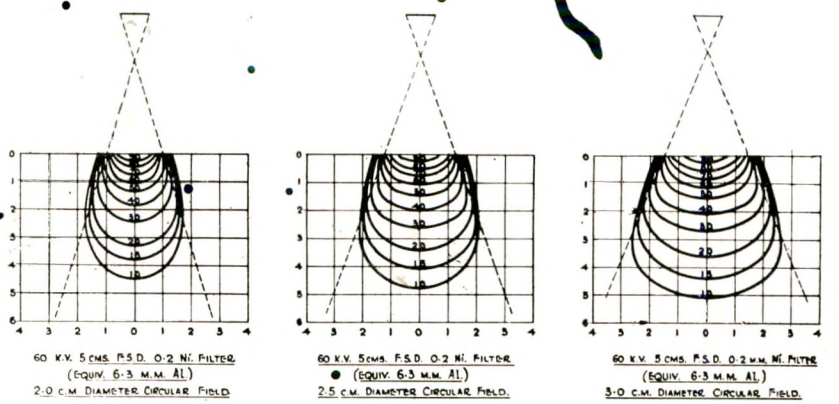


FIG. 7

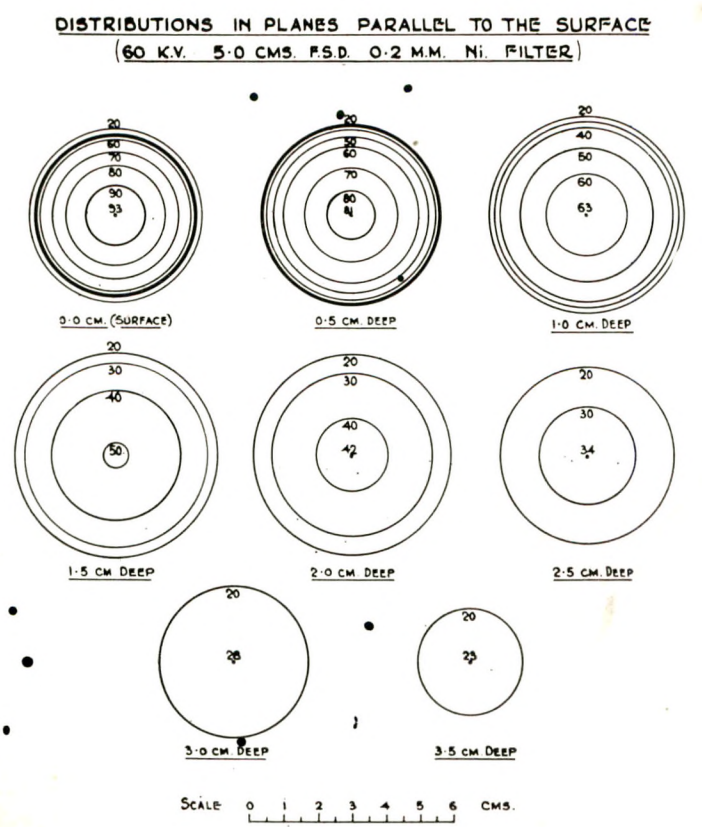


FIG. 8

It is the smallest lesions that patients are apt to consider trivial and these may be adequately treated with a single massive dose in some cases. It is not our experience, however, that patients raise any objection to attending for a few minutes each day for treatment, though this is, no doubt due to the fact that the majority of our patients either come short distances or are admitted to the hospital.

In every case a plan of treatment is worked out for each individual patient. The object is to deliver by fractionated irradiation an adequate tumor dose as evenly as possible to a volume that more than includes the limits of the tumor. Whenever necessary, scale drawings are made and isodose curves applied so as to determine the maximum and minimum tumor doses. These isodose curves have been determined by Dr. W. V. Mayneord and Mr. L. F. Lamerton of the Physics Department and are available for all the depth may be estimated from one isodose curve chart (Fig. 9). Larger tumors are covered with cellophane on which their outlines are drawn. An arrangement of

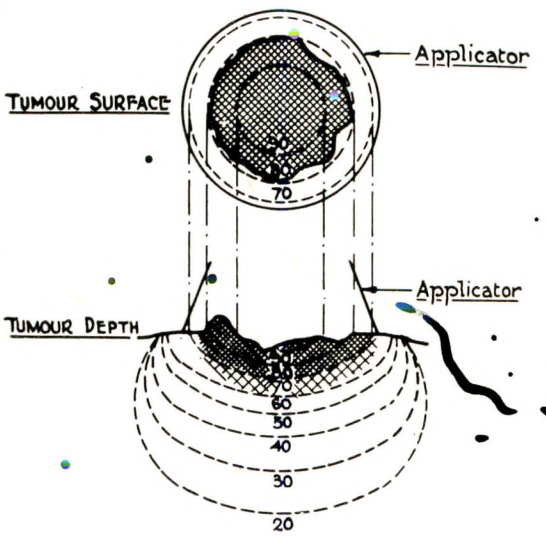


FIG. 9

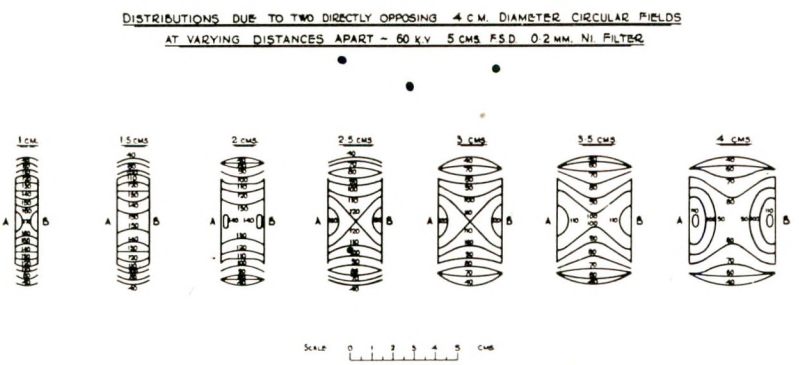
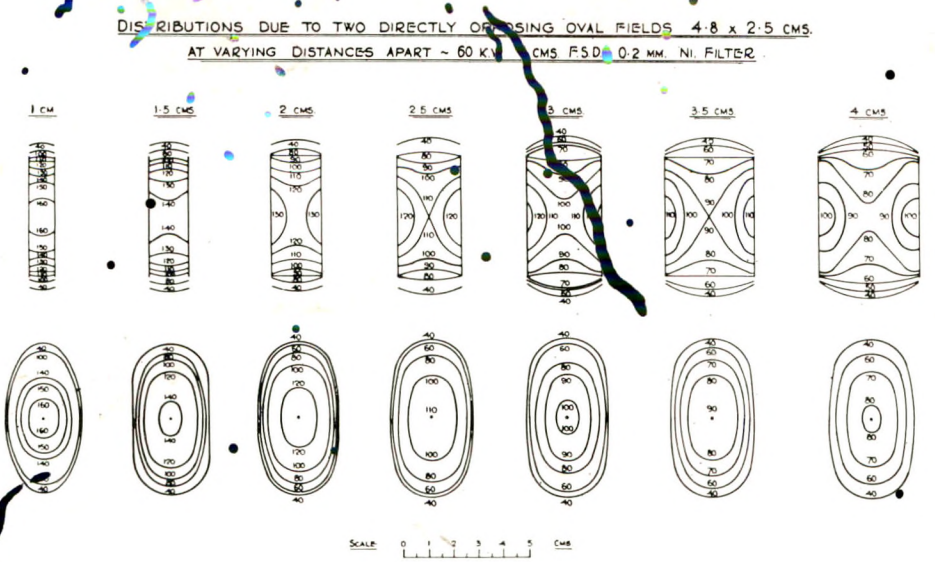


FIG. 10

conditions used (examples are shown in Fig. 6 and 7). From these a variety of distributions have been drawn and can be referred to at any time. As an example one may instance a series of curves showing distributions at varying short distances below the surface (Fig. 8).

Where a tumor can be covered by a single field the dose on the surface and in the fields that cover the tumor is marked on the cellophane, each field being treated and then covered with a lead disc to prevent re-treatment of overlapping surface areas. By varying the arrangement of the fields daily a fairly even surface dose is delivered. The larger tumors that are accessible but extend to a depth of more than 1 to 1.5 cm. are treated with a roentgen tube operating



The upper row of diagrams represents the distributions in the principal plane parallel to the beams in the long axes of the applicators. The lower row of diagrams represents the distributions in the plane at right angles to the beams and mid-way between the fields.

FIG. 11

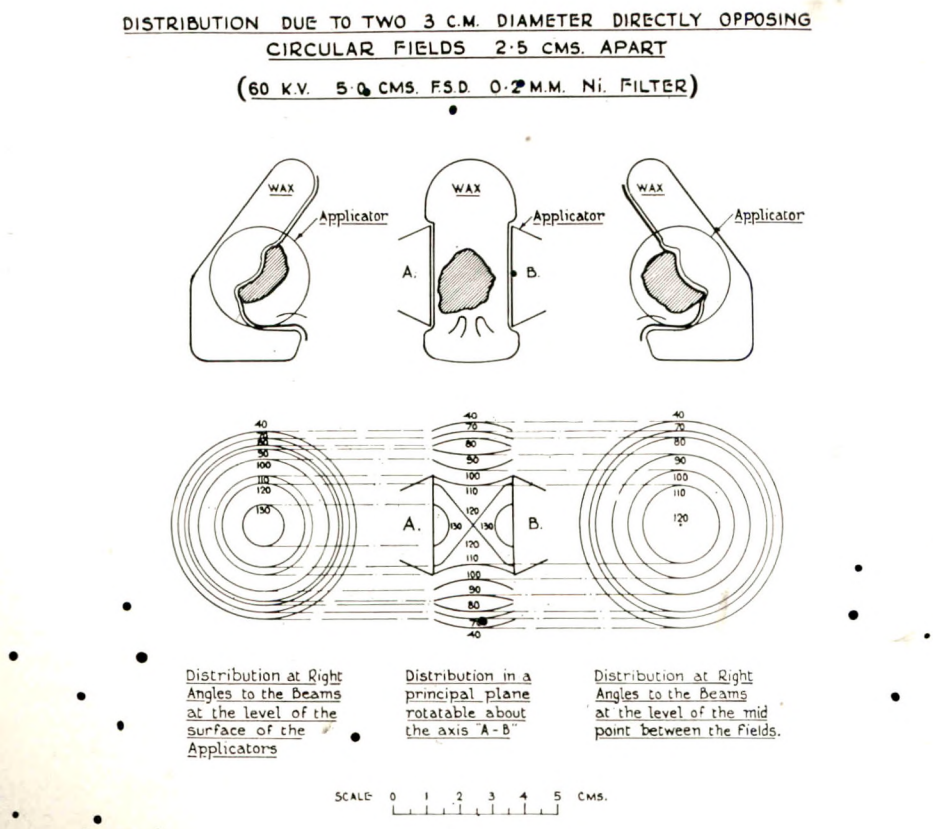


FIG. 12

at 140 kv. with 0.5 mm. Cu filter and a longer focal-skin distance, usually 15 cm. Following the policy of selecting the appropriate distribution for each case a treatment of this kind fills the gap between short distance low voltage and high voltage roentgen therapy. It is in fact middle distance moderate voltage treatment.

Whenever possible, tumors are treated with two directly opposing fields. The distributions due to such fields have been determined for all the varying distances between the fields in common use both for circular (Fig. 10) and for oval fields (Fig. 11). Two opposing fields are used in the treatment of most cases of cancer of the lip, nose, ear, and anterior third of the tongue. In the case of tumors that project irregularly from the surface such as some of those on the nose an impression is taken and a plaster cast made. A strip of wax is moulded on to the plaster cast so that it may fit the patient. With this wax strip in place (Fig. 12), air spaces are eliminated and the tumor is placed in the middle of a homogeneous scattering medium. The position of the fields can be marked on the wax strip and their accurate re-application in the same position assured. In each case the dose given is that which will result in a minimum tumor dose of 4,500–6,500 r for basal and squamous carcinomata in ten to fifteen days.

The average daily dose for single field treatment is 600 r, and a maximum tumor dose of 8,000 r is seldom exceeded.

Eye Protection. Short distance low voltage roentgen therapy is ideally suited for the treatment of tumors in the region of the eyes for the limitation of the irradiation effect itself tends to protect the eyes. When tumors that involve the lids and canthi are to be treated, extra eye protection is necessary. Lead offers the best protection and the voltage employed is not high enough to excite its characteristic radiation. For this purpose we use contact glass or "perspex" lenses to which a 1.0 mm. covering of lead has been applied (Fig. 13). The lead covered lens is inserted under the lids during treatment. These shields transmit less than 0.1 per cent of the radiation; the glass backing offers a non-abrasive surface to the eye, absorbs any β-ray emission from the lead and maintains their shape.



FIG. 13

Access to Tumor by Surgical Exposure. A special branch of short distance low voltage roentgen therapy is the treatment of tumors made accessible by surgical exposure. It is probable that the scope of this type of treatment will be extended considerably in the future. There are two main methods: (1) the tumor may be exposed long enough for a course of fractionated irradiation, and (2) the tumor may be exposed, a single treatment given and the

wound closed immediately, the patient being anesthetized throughout. The first method has been described by Chaoul with reference to the treatment of carcinoma of the rectum. Dr. P. A. Flood at the Royal Cancer Hospital has treated a number of cases of carcinoma of the bladder by the second method. There are certain obvious advantages about a short exposure where a single treatment can be given under aseptic conditions and it is here that the Philips tube is of special value. Being air cooled it is portable and can be taken to the operating theater, and its great radiation output enables the large single doses necessary to be given very rapidly. Unfortunately, the disadvantages of single massive doses remain.

Intracavitary Therapy. It can be seen from the photographs of the roentgen tubes (Fig. 1 and 3) that they are well designed for insertion into cavities. This type of roentgen therapy was in fact originally designed for the treatment of carcinoma of the cervix. A number of tumors in the mouth can also be treated either by contact therapy or by middle distance moderate voltage treatment with special applicators carrying a side eye piece, light and mirror so that the lesion can be seen during the insertion of the tube and accurate positioning ensured.

Table 1 gives some indication of the scope of the type of treatment described. It represents the work done during the first four years with this treatment at the Royal Cancer Hospital.

TABLE 1

PATIENTS TREATED BY SHORT DISTANCE LOW VOLTAGE ROENTGEN THERAPY FROM JULY 1935 TO JULY 1939

Malignant 466		Non-malignant 294		Total 760
Malignant		*Details of Epitheliomata*		
Epitheliomata	156	Scalp, face and neck	42	
Rodent ulcers	134	Lip	31	
Carcinomata of breast (mostly skin recurrences)	131	Trunk and limbs	20	
Malignant melanomata	8	Buccal mucosa, palate and alveolus	20	
Carcinomata of rectum	8	Tongue	17	
Carcinomata of bladder	7	Vulva	11	
Carcinomata of parotid	3	Ear	10	
Carcinoma of lung	1	Penis and scrotum	4	
Miscellaneous	18	Peri-anal	1	
	466		156	

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CONCENTRATION METHOD OF RADIOTHERAPY FOR CANCER OF THE MOUTH, PHARYNX AND LARYNX*

REPORT OF PROGRESS

By MAX CUTLER, M.D.
CHICAGO, ILLINOIS

THIS report of progress presents the results achieved so far by the new method of irradiation called concentration radiotherapy.[1] During the five year period, April, 1938, to April, 1943, 290 cases have been treated, divided as follows:

Cancer of the pharynx and larynx	116 cases
Cancer of the mouth	116 cases
Cancer of the tongue	38 cases
Cancer of the accessory nasal sinuses	20 cases
Total	290 cases

As originally stated, the purpose of these studies is to improve the effectiveness of external irradiation in the more radioresistant types of cancer of the mouth, pharynx, and larynx by systematic controlled clinical research in the basic factors of radiotherapy. Specifically, the following questions are of special interest:

(1) The comparative value of roentgen rays and radium.
(2) The optimum voltage.
(3) The optimum roentgen intensity.
(4) The optimum daily dose, total dose, and treatment time.
(5) The optimum size and number of fields.

The only manner in which these basic questions can be answered is to treat comparable groups of cases under controlled conditions by techniques in which only one factor is varied at a time. This is an immense task and has hitherto not been attempted. The staff of the Chicago Tumor Institute first undertook to study these problems in April, 1938.

After testing numerous variations in the technique of external irradiation, several methods have crystallized which seem to be the most effective. Each technique is applicable to a certain type of lesion as regards location and extent. These techniques are in no way considered as final, but they constitute a definite improvement in the control of the more radioresistant cancers over the former methods. For the purpose of research, it was decided to adopt these several methods and apply them to comparable groups of cancer of the mouth, pharynx, and larynx, varying only one factor at a time. In this manner, it is hoped to evaluate the various basic factors of radiotherapy. For example, three groups of intrinsic carcinoma of the larynx are now treated with roentgen rays and telecurietherapy in which all the factors are the same except that one group is treated with 400 kv. roentgen rays, another with 200 kv. roentgen rays, and a third with telecurietherapy, with the view of ultimately earning the comparative value of the three methods. In a similar manner the factors of roentgen intensity, daily dose, total dose, and total treatment time are being studied.

The following five techniques have been adopted in this research:

(1) *Telecurietherapy, Twelve Treatment Days, Single Constant Portal, Dose 120,000 mg-hr.* This technique is used in lesions of the alveolar ridge and floor of the mouth in which the disease is entirely or mainly unilateral. The following is an example:

CASE 1. Male, aged seventy-three, ulcerated lesion of mucous membrane right upper alveolar ridge extending on to the right cheek. No adenopathy. Biopsy—squamous carcinoma. Treatment—telecurietherapy (10 grams of radium), single right lateral field, 6 by 8 cm. distance 12.5 cm., filtration 1 mm. platinum

* From the Chicago Tumor Institute and the Hines Veterans Hospital aided by a Grant from the National Cancer Institute.

5,000 mg-hr. twice daily for twelve days, total 120,000 mg-hr. between April 7, 1941, and April 19, 1941. At the height of the reaction there developed a pronounced "epithelitis" of the mucous membrane and a mild epidermitis. On June 5, 1941, the lesion had disappeared and there is no evidence of disease at this time, two years and four months after completion of treatment.

Comment. The interesting feature in this case is the use of a large dose over a comparatively short period (twelve days) through a single portal. A similar group of lesions is being treated with roentgen rays in order to compare the results.

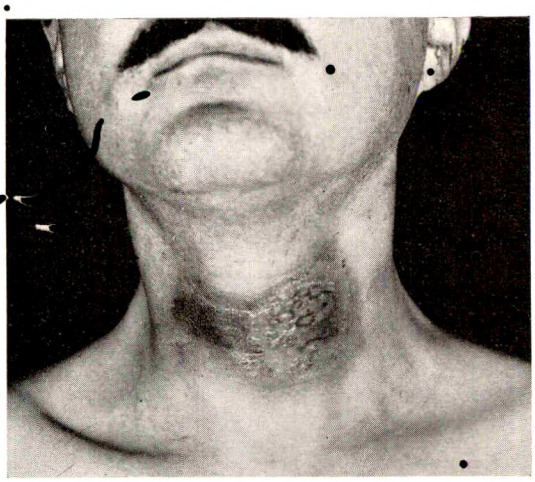


FIG. 1. Case III. Epidermitis first appeared on the twenty-second day from the beginning of treatment and healed ten days later.

(2) *Roentgen Therapy, Twelve Treatment Days, Two Constant Portals, Dose 8,400 r.** This technique is used in lesions of the soft and hard palate. The following is an example:

CASE II. Male, aged fifty-two, ulcerated lesion of left soft palate measuring about 5 by 5 cm. No adenopathy. Biopsy—epidermoid carcinoma transitional cell type.† Treatment—July 7, 1941 to July 19, 1941, 400 kv. roentgen rays, 5 ma., 5 mm. copper, 85 cm. distance, 6 by 6 cm. portals, 5.2 to 6.8 r per minute, 350 r to each side daily and consecutively for twelve treatment days, 4,200 r to each lateral portal, total dose 8,400 r, measured on the skin. Marked "epithelitis" of the mucous membrane but no epidermitis. The lesion disappeared one month after completion of treatment. There is no evidence of recurrence at this time, two years later.

Comment. The interesting feature in this case is the ability to give a very large dose (8,400 r) in a short period of twelve treatment days without untoward local reactions and with restoration of apparently normal conditions in the skin and in the tissues of the tumor bed. The complete disappearance of the lesion in this case is at least partly due to its radiosensitivity since the microscopic structure showed transitional cell carcinoma, but similar results have been observed with adult squamous carcinoma of the palate.

(3) *Roentgen Therapy, Eleven Treatment Days, Single Diminishing Portal, Dose 5,400 r.*‡ This technique is used in intrinsic carcinoma of the larynx affecting the true or false cord or both structures in which for various reasons it is decided to use radiotherapy. The following is an example:

CASE III. Male, aged thirty-eight, ulcerated lesion occupying the left true cord, touching the anterior commissure and almost reaching the posterior commissure. The lesion was rather bulky and extended into the lumen of the larynx, on to the left false cord and subglottically. The left hemilarynx was almost completely fixed. The left arytenoid was partly fixed. The right hemilarynx was normal. Biopsy—squamous carcinoma. Treatment—June 14, 1942 to June 26, 1942, 400 kv. roentgen rays, 5 ma., 5 mm. copper, distance 84 to 115 cm., single left lateral portal 5 by 5 cm. gradually diminished to 3 by 3 cm. Roentgen intensity 4 r per minute to 5.3 r per minute. Two equal treatments daily beginning with 100 r twice daily and increasing to 425 r twice daily, total dose 5,400 r, measured on the skin. After a twelve day interval, the following supple-

* This dose is too strong for some patients and has been reduced to 7,600 r.

† Because of the transitional cell type of lesion, the result in this case cannot be attributed exclusively to the type of irradiation as the lesion may have responded to less intensive treatment.

‡ In some instances an additional 2,000 r has been given to the opposite side about two weeks after the end of the series, as in the case described.

mentary treatment was given to the opposite side of the larynx: 500 r twice daily for two days through a 3 by 3 cm. portal, dose 2,000 r, grand total 7,400 r. The patient developed an "epithelitis" and epidermitis (Fig. 1). The lesion disappeared slowly, no longer being visible five weeks after the end of the main cycle of irradiation. There is no evidence at this time, two years after completion of treatment.

Comment. There are several interesting features about this case. The lesion was operable and probably curable by laryngectomy. It was too advanced for laryngofissure. The patient is a teacher and public speaker, hence he asked for a chance of cure without laryngectomy. The fact that the left hemilarynx was still slightly movable finally led to the decision to try irradiation as the initial procedure. The interesting features of the technique are the use of a single portal, avoidance of using portals that are unnecessarily large, the gradual diminution of the portal with corresponding increase in the daily dose, and the comparatively large total dose in a relatively short treatment period. The appearance of the larynx five weeks after treatment was entirely normal, the mobility of the cords was fully re-established, the voice was normal and the patient regained the 25 pounds he had lost. His morale was exceptionally high as he had been told that cure without laryngectomy was not possible.

(4) *Roentgen Therapy, Interrupted Method, Ten Treatment Days, Two Fields, Diminishing Portals, Dose 7,700 r.** This technique is used in more advanced intrinsic carcinoma of the larynx in which the advantages of the therapeutic test are desired. In certain operable intrinsic lesions this method of interrupted treatment permits one to estimate the probable radiosensitivity of the lesion before the second phase of the irradiation is given. In this manner, the second cycle can be omitted if the lesion appears to be relatively radioresistant and laryngectomy can be resorted to. The following is an example:

* A third cycle consisting of 2,000 r in two days is sometimes added as in the case reported here.

CASE IV. Male, aged forty-three, contributor, and increasing hoarseness for one year, pain on swallowing for three months. Examination—right true and false cords are the seat of a large ulcerated mass projecting into the lumen of the larynx with almost complete fixation of the right hemilarynx (Fig. 2). Biopsy—squamous carcinoma. For six days (February 24, 1941 to March 1, 1941) the patient received two treatments daily as follows: 400 kv. roentgen rays, 5 mm. copper filter, 85 cm. distance, 5 ma., portals 30 to 20 sq. cm. Doses: first day 400 r, second day 500 r, third day 550 r, fourth day 650 r, fifth day 700 r, sixth day 800 r, total dose 3,600 r, measured on the skin. Roentgen intensity varied between 6.3 r per minute and 8.9 r per minute. The first cycle of treatment

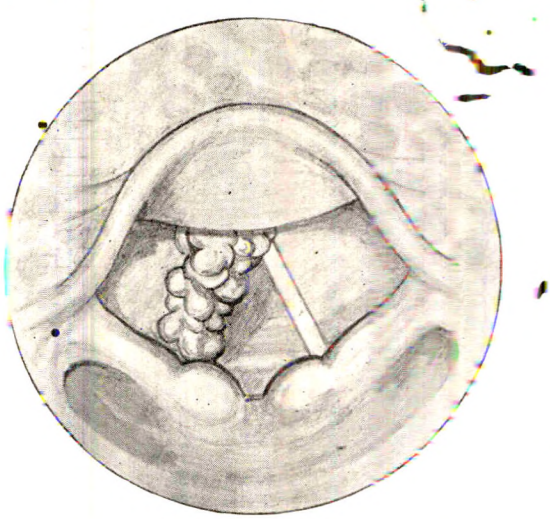


FIG. 2. Case IV. Squamous carcinoma of the right true and false cords. (Drawing by D. Holsey, M.D.)

was given over the right side of the larynx. On March 12, 1941, eleven days after completion of the first roentgen-ray cycle, the lesion showed the first sign of regression and there was more mobility of the right hemilarynx. Two days later, the thirteenth day after the last treatment, there was marked further regression and increased mobility.

Because of this pronounced improvement, it was decided to administer the second cycle of roentgen-ray treatments. The interval between the two cycles was twelve days. The second cycle began March 14, 1941, and finished March 17, 1941, with the following factors: 400 kv. roentgen rays, 5 mm. copper, 85 cm. distance, 5 ma., portals 30 sq. cm. to 12 sq. cm.

doses: first day 850 r, second day 950 r, third day, 1,050 r, fourth day, 1,150 r, two treatments daily, total dose 4,000 r, measured on the skin. The total dose during the second cycle was given over the left side of the larynx. The first cycle was 3,600 r and the second cycle 4,000 r, making a total of 7,600 r for the two cycles during ten treatment days.

The maximum reaction on the skin consisted of a deep pigmentation. The maximum mucous membrane reaction was a very mild "epithelitis." The mobility of the right hemilarynx was restored to normal on April 4, 1941, eighteen days after the completion of the second cycle of irradiation. The lesion continued to regress and finally disappeared on April 25, 1941, thirty-eight days after the last treatment of the second cycle, but there remained a small, irregular, non-ulcerated nodule about 6 mm. in diameter situated on the posterior extremity of the right true cord. On May 9, 1941, fifty-three days after the last treatment, it was decided to administer a third course of irradiation. It was not possible to decide clinically whether or not this nodule contained a remnant of carcinoma and it was felt that a biopsy should not be done. From previous experience, it seemed that a third course of irradiation could be given with safety. Consequently, the third course was administered in two days as follows: Field over right side of larynx, 400 kv. roentgen rays, 5 mm. copper, 80 cm. distance, 7 sq. cm. portal, 7.4 r to 9.8 r per minute, 500 r twice daily for two days or a total of 2,000 r, measured on the skin. The grand total for the three cycles of irradiation was 9,600 r. The last treatment was given on May 10, 1941. The nodule regressed slowly and finally resolved into a small fibrotic thickening about 3 mm. in diameter which has remained stationary for three years. It is obvious that this is an area of scar tissue. On April 29, 1942, the patient had gained about 20 pounds in weight, the voice was normal, and the right hemilarynx was normal in appearance and mobility.

Comment. There are several significant features about this case. The most important is the disappearance of an extensive intrinsic squamous carcinoma of the larynx with almost complete fixation of the hemilarynx, with freedom from recurrence for three years. Several features of the treatment are interesting. The large total dose (9,600 r) administered in only twelve treatment days was possible with very little reaction in the skin and mucous membrane. The division of the treatment into three cycles with two intervals afforded an opportunity to observe the response of the lesion and plan further treatment accordingly. Finally, this case illustrates that it is practical to test the radiosensitivity of a lesion with a partial irradiation and use such information as a guide to further treatment.

For some patients this treatment is too intense and has resulted in radionecrosis, hence the doses have been reduced about 20 per cent.

(5) *Roentgen Therapy, Eighteen Treatment Days, Single Diminishing Portal, Dose 6,500 r.* This technique is used in extrinsic carcinomas of the larynx (pyriform fossa, aryepiglottic fold, epiglottis). The following is an example:

CASE V. Male, aged fifty-seven. There is an extensive swelling over the region of the thyroid and cricoid obliterating the normal contour of the anterior surface of the neck (Fig. 3*A*). There is a marked swelling and ulceration in the region of the right true and false cords, extending to the subglottic region. The right arytenoid is enlarged, the right hemilarynx is partly fixed. There is no cervical adenopathy. Biopsy showed squamous carcinoma. Soft tissue roentgenography of the larynx disclosed extensive destruction of the cricoid and thyroid cartilages. Treatment was given between July 7 and July 30, 1938, over a period of twenty-one treatment days, one treatment daily through a single right lateral field, 400 kv., 5 ma. 8 mm. copper, distance varied between 65 cm. and 90 cm., size of portal varied between 8 by 10 cm. and 3 by 4 cm., daily dose increased gradually from 100 r to 700 r, measured on the skin, roentgen intensity varied between 4.1 r per minute and 7.5 r per minute. Total dose 6,500 r. The tumor regressed rapidly during the first few days of treatment. Approximately one week after beginning treatment, an abscess pointed over the thyroid region which was incised and evacuated. This resulted in marked relief. The weight increased and the general condition improved. On October 6, 1938, about ten weeks after the comple-

tion of treatment, there was no definite evidence of disease, the general condition was excellent, and the patient had no complaints. Subsequent examination of soft tissue roentgenograms of the larynx disclosed a remarkable restoration of the cartilages to what seems to be a normal state. There is no sign of recurrence at this time, about six years after completion of treatment (Fig. 3*B*).

Comment. This technique* is the most effective so far observed for the more extensive intrinsic carcinomas and for extrinsic carcinomas of the larynx. When the cervical adenopathy has been too extensive to be included in the fields, an additional cycle of treatment has been given to such areas as could not be included in the first cycle of treatment.



A *B*

Fig. 3. Case v. *A*, extensive carcinoma of the right hemilarynx with perforation through the thyroid and cricoid cartilages and obliteration of the normal contour of the neck before treatment. *B*, five years after treatment showing restoration of the normal contour of the neck.

DISCUSSION

The basis of the method of concentration is the use of large daily doses over a comparatively short treatment period (ten to eighteen days). The total dose is sufficient to produce an "epithelitis" and in some instances an epidermitis and there is reason for believing that the total dose under the given conditions constitutes a maximum irradiation. It is by no means certain that all the factors are the most favorable, except that *the increase in the daily dose and diminution in the total treatment period has caused regression, disappearance, and apparent cures of lesions that have failed to respond to the former divided dose technique.* There is reason to expect that the technique of gradually increasing the daily dose and diminishing the size of the field is of advantage but this principle cannot be adequately supported on theoretical grounds and the data at hand are not sufficient to settle the question definitely at this time. The idea of delivering the largest doses through the smallest fields in the last few days is intriguing, and for certain types of carcinomas, as in the larynx, this technique appears to be especially effective.

Before 1922 surgery was the only form of treatment for operable carcinomas of the larynx. The first successful results of radiotherapy were reported by Regaud, Coutard and Hautant[5] who presented 6 inoperable cases treated successfully with roentgen therapy. In 1938 Jackson and Jackson[2] stated their belief that the future will prob-

* This was the first case treated by this method and the treatment time was twenty-three days. The treatment time has now been reduced to eighteen days and two treatments are used daily instead of one. The total dose remains the same.

ably see a progressive decrease in the relative number of laryngectomies and an increase in the number of patients treated with irradiation. Similar views have been expressed by Quick[4] and others. Lenz[3] has published favorable results of roentgen therapy in cancer of the larynx.

As long as radiotherapy was limited to inoperable lesions, the problem of choosing between operation and irradiation was relatively simple. Now that irradiation is considered to be the method of choice in certain operable lesions, the problem has become much more difficult. The responsibility of advising radiotherapy instead of laryngectomy in certain conditions in which the opposing factors appear to be equally balanced is indeed a serious matter. The knowledge that if radiotherapy fails, laryngectomy may still be performed with safety is of considerable help. As experience accumulates, the decision becomes less difficult. Intrinsic lesions of the true vocal cord

TABLE I

CARCINOMA OF LARYNX
(True Cord)

Early—cord movable	35%*
Border line—cord partly movable	35%
Advanced—cord fixed	30%

Year	Cases Treated	Alive at End of				
		1939	1940	1941	1942	May 1943
1938	7	5	5	5	5	4
1939	7	7	5	5	5	5†
1940	9		7	6	5	5
1941	21			21	17	15
1942	12				12	10‡
1943	1					1
	57					40

* These figures represent the percentages of the total number of cases included in the table.
† Laryngectomy followed radiotherapy in 1 case.
‡ Cardiac death, no evidence of cancer in 1 case; laryngectomy followed radiotherapy in 2 cases.

TABLE Ia

CARCINOMA OF LARYNX
(True Cord—Movable and Partly Fixed)

Year	Cases Treated	Alive at End of				
		1939	1940	1941	1942	May 1943
1938	7	6	5	5	4	4
1939	4	4	3	3	3	3
1940	6		5	5	4	4
1941	13			13	11	11
1042	9				9	9
1943	2					2
	41					33

TABLE Ib

CARCINOMA OF LARYNX
(True Cord, Movable)

Year	Cases Treated	Living and Well	Died in		
			1939	1940	1942
1938	2		1		1*
1939	3	2		1	
1940	3	3			
1941	7	7			
1942	3	3			
1943	2	2			
	20	17			

Seventeen living and well, average length of time since treatment—25 months.

* Died of cerebral metastasis—primary lesion controlled.

TABLE II

CARCINOMA OF LARYNX AND HYPOPHARYNX
(Epiglottis, False Cord, Pyriform Fossa)

Year	Treated	Alive at End of				
		1939	1940	1941	1942	May 1943
1938	11	5	3	3	2	2
1939	6	6	4	3	3	3
1940	4		3	2	2	2
1941	18			18	14	10*
1942	18				18	17
1943	1					1
	58					35

Sixty-two per cent of lesions were advanced and associated with cervical adenopathy.

* Laryngectomy following radiotherapy in 1 case.

with subglottic extension and complete fixation have failed to respond to the most intensive radiotherapy; consequently laryngectomy is performed in these cases if there are no contraindications. When the fixation of the vocal cord is only partial, irradiation is usually the initial procedure.

TABLE III

CARCINOMA OF MOUTH, OROPHARYNX AND NASOPHARYNX

(Excluding Tongue)

Early	10.5%
Advanced	34.5%
Very advanced	55%

Year	Cases Treated	Alive at End of 1939	1940	1941	1942	May 1943
1938	13	6	5	5	3	3
1939	15	9	5	5	4	2
1940	17		15	11	8	8
1941	35			28	22	21*
1942	27				23	21
1943	9					9
	116					64

* Excision of remnant in 1 case; neck dissection after radiotherapy in 1 case. Five cases died of causes other than cancer (clinically free of disease).

TABLE IV

CARCINOMA OF TONGUE

Early	13%
Advanced	39%
Very advanced (with adenopathy)	48%

Year	Cases Treated	Alive at End of 1939	1940	1941	1942	May 1943
1938	5	0	0	0	0	0
1939	2	2	0	0	0	0
1940	8		6	4	2	2
1941	16			14	10	10*
1942	6				5	4†
1943	1					1
	38					17

* Hemiglossectomy following radiotherapy in 1 case.

† Neck dissection and hemiglossectomy following radiotherapy in 1 case.

TABLE V

CARCINOMA OF ACCESSORY NASAL SINUSES

Year	Cases Treated	Alive at End of 1939	1940	1941	1942	May 1943
1938	6	4	4	4	3	3
1939	2	1	1	1	1	1
1940	0					
1941	8			5	3	3
1942	3				3	3
1943	1					1
	20					11

The accompanying tables give the results of treatment of the 290 cases which form the subject of this research.

SUMMARY

(1) A method of radiotherapy has been evolved which is called the concentration method. Roentgen rays or radium may be used. This method has resulted in the eradication of the more radioresistant carcinomas of the mouth, pharynx, and larynx which have failed to respond to the previous methods of external irradiation.

(2) As a result of this development, the initial disappearance and apparent cures have been observed in a group of intrinsic squamous carcinomas of the larynx which are generally regarded as radioresistant and for which surgery is usually claimed to be the only effective method of treatment. In many of these cases the only alternative to radiotherapy was complete laryngectomy.

(3) Although it is necessary to treat more cases and wait still longer in order to be more certain of the permanency of the results, it is already clear that the surgical attitude toward the treatment of some forms of intrinsic cancer of the larynx must, on the basis of these results, be altered.

(4) A radiotherapeutic test has been developed for certain border line cases of intrinsic cancer of the larynx in which a decision between radiotherapy and laryngectomy is difficult. Approximately half the

full dose of treatment is given in six days as a test of the sensitivity of the lesion. This is followed by an interval of fifteen days of observation. The degree of regression of the lesion at the end of the twenty-one days can be used as a reasonably accurate index of the radiocurability of the lesion. If the response to the first course of treatment is adequate, the second course is administered. If the response has been inadequate, laryngectomy may be performed with safety. This method has been a great help in certain difficult border line cases and gives the patient a chance of cure with preservation of his voice in the event the lesion proves to be sensitive to irradiation. It also does not eliminate his chances of cure by laryngectomy if the lesion proves to be radioresistant.

(5) Five different techniques of irradiation have been developed under the method of concentration. Each is applicable to a certain type of cancer of the mouth, pharynx, and larynx.

(6) The results in radioresistant forms of cancer of the mouth have been improved by the method of concentration.

(7) A study of the comparative value of 220 kv. roentgen rays, 400 kv. roentgen rays, and radium has been started. The study has not proceeded far enough to justify an opinion at this time.

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RADIUM DOSAGE FOR LINEAR SOURCES
TABLE AND NOMOGRAM*

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IN a previous article,[3] data on dosage for linear sources of radium, calculated by the method of Laurence,[1,2] were presented in graphical form. Because of difficulties in graph reading, the data are here presented in two simpler forms—a table and a nomogram.

Table 1 gives directly the dose in gamma roentgens (r_γ) per milligram-hour along the "end" axis, "in air," for filtrations equivalent to 0.5 mm. platinum. Other filtrations and tissue absorption are considered later. By "end" axis is meant the line perpendicular to the linear source at its end, e.g., *BN* in Figure 1 and not the line which would represent the prolongation of the linear source. In reality, the "end" axis is a plane, but appears in the diagram as a line. "Distance" refers to the distance measured along this or other perpendicular axes in centimeters. The distribution of the radium salt in the linear source is assumed to be uniform.

The value 9.0 was used as the number of gamma roentgens from a 1 milligram point source of unfiltered radium element at 1 centimeter in 1 hour.

A dosage table for points along an end axis is valuable because it can be used to obtain the dose at all points. This arises from the fact that a perpendicular line from any given point to a linear source may be considered an end axis for the two linear segmental sources into which the original or true linear source is divided by this line. The following examples will make this clearer and illustrate the use of the method. An active length of 4 cm. and a treatment distance of 2 cm. are arbitrary figures selected only for convenience of demonstration.

Assume a linear source of active length 4 cm. (*AB* of Fig. 1, 2 and 3) and filtration equivalent to 0.5 mm. Pt:

Example 1. Find the dose per milligram-hour 2 cm. away from the linear source along the end axis, i.e., at point *N* (Fig. 1).

The dose at point *N* is read directly from Table 1 as 1.10 r_γ per milligram-hour by following the vertical line under active length 4 and a horizontal line from distance 2.

Example 2. Find the dose per milligram-hour 2 cm. away from the linear source along the "central" axis, i.e., at *M* (Fig. 1). A central axis is a line perpendicular to the linear source at its center.

The line *OM* may be assumed to be the

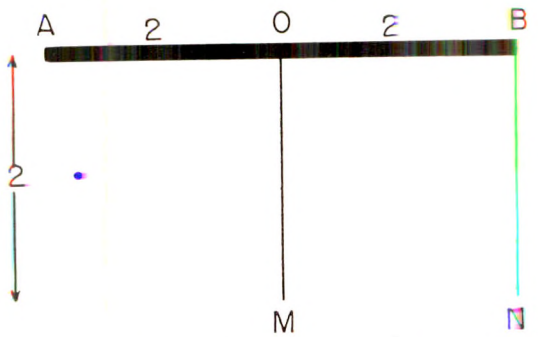


FIG. 1. *AB* is the active length of the linear source. *BN* is the end axis; *OM* is the central axis. The dose at point *N* is calculated in example 1. The dose at point *M* is calculated in example 2.

end axis for the two segmental linear sources *AO* and *BO*, each of which contributes 0.5 milligram-hour if the total contribution from *AB* is 1 milligram-hour.

The dose at *M* from the linear segment $AO = 1.62/2 = 0.81$ r_γ for 0.5 milligram-hour (1.62 is the dose per milligram-hour from the linear source *AO* obtained from Table 1. This figure must be divided by 2 since *AO* contributes only one-half of one milligram-hour).

The dose at *M* from *BO* is the same as the dose from *AO*. Therefore the dose at *N* from both *AO* and *BO*, i.e. from *AB*, equals

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twice the dose due to AO alone for twice the number of milligram-hours from AO alone, or:

Dose due to $AB = 2 \times 0.81$ or $2 \times 1.62/2 = 1.62$ r_γ for 1 mg-hr.

It turns out that we have both divided and multiplied the dose from AO by 2 or that the dose per milligram-hour along the central axis of a linear source 4 cm. long is equal to the dose per milligram-hour

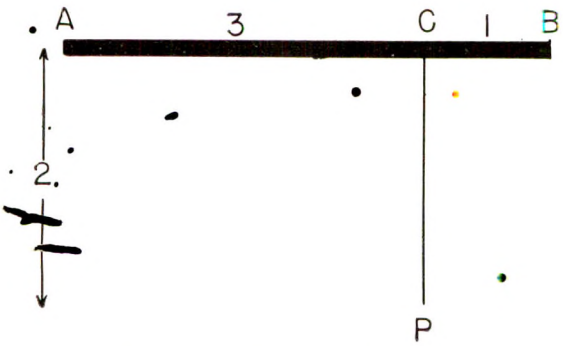


FIG. 2. AB is the active length of the linear source; CP is the perpendicular axis through a point 1 cm. from the end B and 3 cm. from the end A. The dose at point P is calculated in example 3.

along the end axis of a source 2 cm. long at the same treatment distance. This is the general rule for points on a central axis. Therefore, one can find the dose per milligram-hour at any point along a central axis by looking up in the table under one-half the active length instead of the full active length. Calculations for this type of case are therefore unnecessary and the table is used directly.

Example 3. Find the dose per milligram-hour 2 cm. away from the linear source along an axis 3 cm. from one end and 1 cm. from the other end, i.e., at P (Fig. 2). The two segmental linear sources are AC and BC (Fig. 2).

For purposes of calculation in cases of this type, it is usually simpler to assume that the number of milligram-hours represented by the linear source itself and each of its segments is equal to the number of centimeters in its length instead of assuming that the linear source represents 1 mg-hr. and each segment a fraction thereof depending on its length. This assumption is possible because the linear density of the radium salt in the capsule or needle is uniform and therefore the number of milligram-hours contributed by each segment is proportional to its length.

Assume that the segment AC, being 3 cm. long, contributes 3 mg-hr. The segment BC, 1 cm. long, contributes 1 mg-hr.

From the table, we find that the dose per milligram-hour from the segment AC (active length 3 cm.) at the point P (distance 2 cm.) is 1.34 r_γ. For 3 mg-hr., this figure must be multiplied by 3:

Dose at P from AC for 3 mg-hr. $= 1.34 \times 3 = 4.02$ r_γ.

Similarly, the dose at P from BC for 1 mg-hr. $= 1.88 \times 1 = 1.88$ r_γ.

When we add the dose from AC (for 3 mg-hr. of AC) to the dose from BC (for 1 mg-hr. of BC), we obtain the dose from

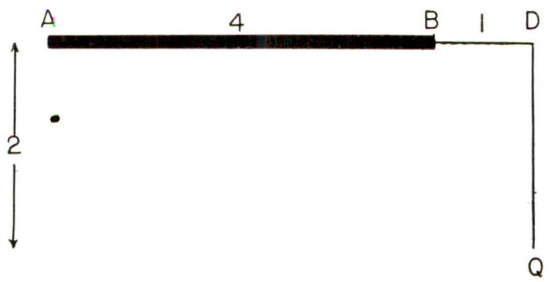


FIG. 3. AB is the active length of the linear source; DQ is the perpendicular axis through a point 1 cm. beyond the end B. The dose at point Q is calculated in example 4.

AB, i.e., from the true linear source (for 4 mg-hr. of AB):

Dose at P from AB for 4 mg-hr. $= 4.02 + 1.88 = 5.90$ r_γ. The figure 4 mg-hr. is of course the sum of 3 and 1 mg-hr. and is also equal to the total active length in centimeters.

Since we know the dose for 4 mg-hr., we can easily find the dose for any other number of milligram-hours. For 1 mg-hr., the dose $= 5.90/4 = 1.48$ r_γ.

Example 4. Find the dose per milligram-hour 2 cm. away from the linear source along an axis 1 cm. beyond the end of the linear source, i.e., at Q (Fig. 3). In this

case, the two "segments" are AD, 5 cm. long, and BD, 1 cm. long. (The "segment" AD is actually larger than the linear source itself, but the term segment is still used by analogy with the previous case.) We assume that AD contributes 5 mg-hr. and BD 1 mg-hr. Clearly, in this example, the dose from BD must be subtracted from the dose due to AD in order to obtain the dose from AB, the true linear source.

Dose at Q from AD for 5 mg-hr. $= .94 \times 5 = 4.70$ r_γ.

Dose at Q from BD for 1 mg-hr. $= 1.88 \times 1 = 1.88$ r_γ.

Dose at Q from AB for 4 mg-hr. $= 4.70 - 1.88 = 2.82$ r_γ. The figure 4 is the difference between 5 and 1 mg-hr. and is equal to the number of centimeters in the total or true active length.

Dose at Q per milligram-hour of AB $= 2.82/4 = 0.71$ r_γ.

The data included in the table may also be put in the form of a nomogram. This is an alignment chart (Fig. 4) consisting of two vertical scales and a central oblique

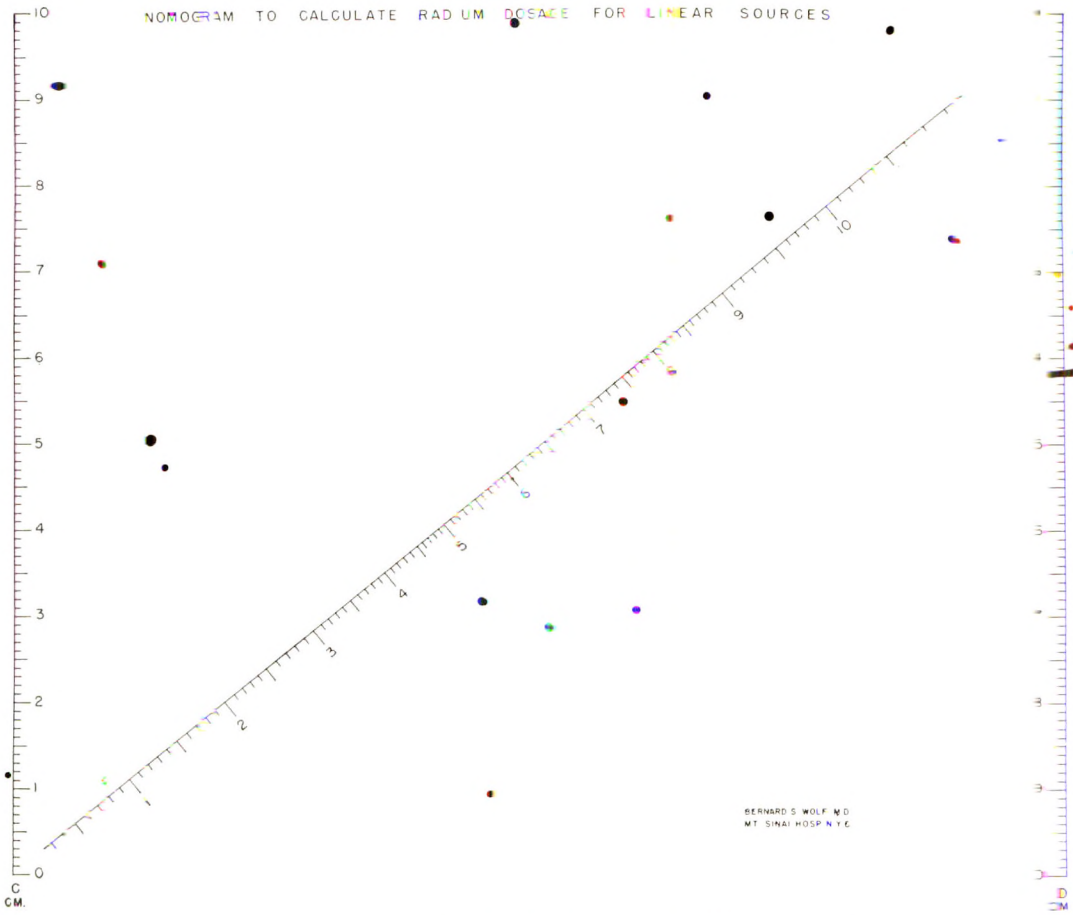


Fig. 4. Nomogram to calculate dosage for linear sources. For explanation, see text.

scale. The vertical scale on the left is called the C scale; the vertical scale on the right, the D scale, and the central oblique scale, the F scale. The advantages of a nomogram are that it is compact and that all intermediate values are easily obtained. Any linear source is looked upon as consisting of two separate linear sources or segments, as described in the previous examples. Each of these two segments is treated separately in succession by the nomogram in exactly the same way. On the left vertical scale (C scale), one finds the active length of the

segment. On the right vertical scale (*D* scale), one finds the treatment distance. Then, a straight edge—e.g., a ruler, preferably transparent, or a piece of string stretched tightly—is placed across the chart through the two selected points on the *C* and *D* scales. This straight edge intersects the central oblique scale at a point which gives an *F* figure for each of the two segments. The two *F* figures thus obtained are then added—or subtracted if the point in question lies beyond the end of the linear source (as in *Example 4* above). One further step is necessary when the nomogram is used. Because of the form of the fundamental equation for dosage from linear sources, the result of the addition or subtraction of the two *F* figures must be divided by the total active length and by the treatment distance before the dose per milligram-hour is obtained.

To demonstrate the use of the nomogram, the same examples (in different order) will be solved.

Example 3 (Fig. 5). For the 3 cm. segment, we connect 3 (active length in centimeters) on the *C* scale with 2 (treatment distance in centimeters) on the *D* scale and obtain 7.8 as the first *F* value, or $F_1 = 7.8$.

For the 1 cm. segment, we connect 1 on the *C* scale with 2 on the *D* scale and obtain

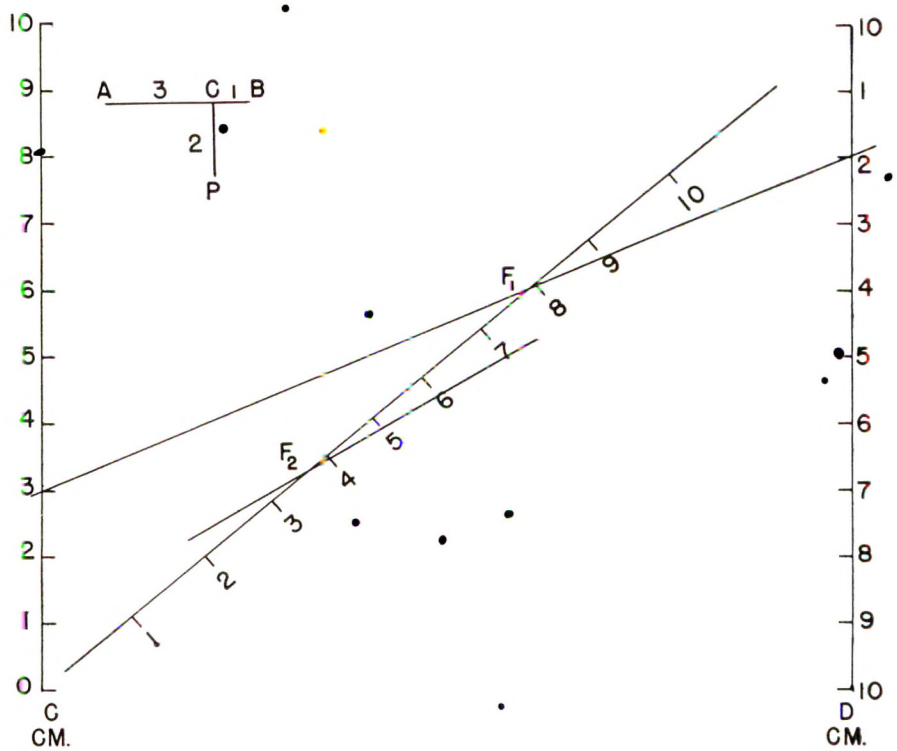


FIG. 5. Example of use of nomogram. The straight edge for the F_2 value connecting 1 on the left (*C*) scale with 2 on the right (*D*) scale is only partially shown. F_1 is derived from segment *AC*; F_2 is derived from segment *BC*.

$F_2 = 3.6$. The two *F* figures are then added:

$$F_1 + F_2 = 11.4$$

This figure must be divided by 4 (active length of original or true linear source) and by 2 (treatment distance) to obtain the dose per milligram-hour:

Dose at *P* from *AB* per milligram-hour

$$= \frac{11.4}{4 \times 2} = \frac{11.4}{8} = 1.43 \text{ r}_\gamma$$

Example 4. One segment is 5 cm. long, the other 1 cm. long.

TABLE I

ROENTGENS PER MILLIGRAM-HOUR ALONG "END" AXIS; 0.5 MM. Pt

Distance in Centimeters	Active Length in Centimeters								
	.3	.4	.5	.6	.7	.8	.9	1.0	1.1
.3	72	63	55	49	44	40	36	33	31
.4	44	41	37	34	31	28.4	26.2	24.3	22.5
.5	29.5	28.5	26.3	23.3	21.7	20.0	18.5	17.2	16.2
.6	20.5	19.5	18.5	17.3	16.2	15.2	14.3	13.5	12.7
.7	15.9	15.6	15.0	14.0	13.0	12.2	11.5	10.8	10.3
.8	11.7	11.5	11.3	10.3	10.3	9.8	9.4	8.9	8.5
.9	9.3	9.2	9.0	8.3	8.5	8.2	7.8	7.5	7.2
1.0	7.5	7.5	7.5	7.4	7.2	6.9	6.6	6.4	6.2
1.2	5.3	5.3	5.3	5.2	5.1	5.0	4.8	4.7	4.5
1.4	3.9	3.9	3.9	3.9	3.8	3.7	3.6	3.6	3.5
1.6	3.0	3.0	3.0	2.95	2.92	2.88	2.84	2.80	2.75
1.8	2.40	2.40	2.40	2.38	2.36	2.33	2.30	2.27	2.25
2.0	1.95	1.95	1.95	1.94	1.93	1.91	1.90	1.88	1.87
2.2	1.62	1.62	1.62	1.62	1.59	1.58	1.56	1.55	1.54
2.4	1.36	1.36	1.36	1.36	1.34	1.33	1.32	1.30	1.29
2.6	1.15	1.16	1.16	1.15	1.15	1.14	1.13	1.12	1.11
2.8	1.00	1.00	1.00	1.00	1.00	1.00	.99	.98	.98
3.0	.83	.88	.88	.88	.88	.88	.88	.87	.86
3.2	.79	.79	.79	.79	.79	.78	.78	.77	.76
3.4	.71	.71	.71	.71	.71	.69	.69	.68	.68
3.6	.64	.64	.64	.64	.64	.63	.62	.61	.61
3.8	.55	.56	.56	.56	.56	.56	.55	.55	.54
4.0	.50	.50	.50	.50	.50	.50	.49	.49	.49
4.5	.39	.39	.39	.39	.39	.39	.39	.39	.39
5.0	.33	.33	.33	.33	.33	.33	.33	.32	.32
5.5	.270	.270	.270	.270	.270	.270	.270	.269	.268
6.0	.225	.225	.225	.225	.225	.225	.225	.224	.223
6.5	.185	.185	.185	.185	.185	.185	.185	.185	.185
7.0	.165	.165	.165	.165	.165	.165	.165	.165	.165
7.5	.140	.140	.140	.140	.140	.140	.140	.140	.140
8.0	.126	.126	.126	.126	.126	.126	.126	.126	.126

	1.2	1.3	1.4	1.5	1.6	1.7	1.8	1.9	2.0
.3	28.5	26.8	25.0	23.5	22.3	21.0	20.0	19.0	18.0
.4	21.1	19.8	18.5	17.5	16.5	15.6	14.9	14.1	13.5
.5	15.2	14.3	13.5	12.8	12.2	11.7	11.2	10.8	10.3
.6	12.0	11.4	10.8	10.3	9.8	9.4	9.0	8.6	8.2
.7	9.7	9.2	8.8	8.4	8.0	7.7	7.4	7.2	6.9
.8	8.1	7.8	7.4	7.1	6.8	6.6	6.4	6.0	5.8
.9	6.9	6.6	6.3	6.0	5.8	5.6	5.4	5.2	5.0
1.0	5.9	5.7	5.5	5.3	5.1	4.9	4.7	4.6	4.4

TABLE I—*Cont.*

Distance in Centimeters	Active Length in Centimeters								
	1.2	1.3	1.4	1.5	1.6	1.7	1.8	1.9	2.0
1.2	4.4	4.3	4.2	4.0	3.9	3.8	3.7	3.5	3.4
1.4	3.4	3.3	3.2	3.1	3.0	2.93	2.85	2.76	2.70
1.6	2.70	2.65	2.60	2.55	2.49	2.43	2.37	2.32	2.27
1.8	2.22	2.18	2.14	2.10	2.06	2.02	1.97	1.93	1.89
2.0	1.86	1.84	1.81	1.78	1.75	1.72	1.69	1.65	1.62
2.2	1.52	1.50	1.48	1.46	1.44	1.42	1.40	1.38	1.35
2.4	1.28	1.27	1.26	1.25	1.24	1.22	1.21	1.19	1.18
2.6	1.10	1.09	1.08	1.07	1.06	1.05	1.04	1.03	1.02
2.8	.97	.96	.95	.94	.93	.92	.92	.90	.90
3.0	.86	.84	.84	.83	.82	.81	.80	.80	.79
3.2	.76	.75	.75	.74	.73	.72	.72	.71	.71
3.4	.68	.67	.67	.66	.56	.65	.64	.64	.63
3.6	.60	.60	.60	.59	.59	.58	.58	.57	.57
3.8	.54	.53	.53	.53	.52	.52	.51	.51	.51
4.0	.49	.48	.48	.48	.47	.47	.46	.46	.46
4.5	.39	.39	.39	.39	.39	.39	.39	.38	.38
5.0	.32	.32	.32	.32	.31	.31	.31	.31	.31
5.5	.267	.266	.265	.265	.264	.263	.262	.261	.260
6.0	.222	.221	.220	.220	.220	.219	.218	.217	.216
6.5	.185	.185	.185	.184	.184	.184	.183	.183	.182
7.0	.164	.164	.164	.164	.163	.163	.163	.162	.162
7.5	.140	.140	.140	.140	.140	.140	.140	.140	.140
8.0	.126	.126	.126	.126	.126	.126	.126	.125	.125
	2.1	2.2	2.3	2.4	2.5	2.6	2.7	2.8	2.9
.3	17.2	16.4	15.7	15.1	14.5	14.0	13.5	13.0	12.5
.4	12.9	12.3	11.7	11.3	10.9	10.5	10.1	9.7	9.4
.5	9.9	9.5	9.1	8.8	8.5	8.2	7.9	7.6	7.4
.6	8.0	7.7	7.4	7.2	6.9	6.6	6.4	6.2	6.0
.7	6.6	6.4	6.2	6.0	5.8	5.6	5.4	5.2	5.1
.8	5.6	5.5	5.3	5.1	4.9	4.8	4.7	4.5	4.4
.9	4.8	4.7	4.5	4.4	4.3	4.1	4.0	3.9	3.8
1.0	4.3	4.2	4.0	3.9	3.8	3.7	3.6	3.5	3.4
1.2	3.3	3.3	3.2	3.1	2.99	2.90	2.83	2.75	2.68
1.4	2.63	2.56	2.50	2.44	2.38	2.32	2.27	2.22	2.17
1.6	2.21	2.15	2.10	2.05	2.00	1.95	1.91	1.86	1.83
1.8	1.85	1.81	1.77	1.73	1.69	1.65	1.62	1.59	1.56
2.0	1.59	1.56	1.53	1.50	1.47	1.45	1.42	1.39	1.37
2.2	1.33	1.31	1.29	1.27	1.25	1.23	1.21	1.19	1.17
2.4	1.16	1.14	1.13	1.11	1.09	1.07	1.05	1.04	1.02
2.6	1.01	1.00	.98	.98	.96	.94	.93	.92	.90
2.8	.89	.88	.87	.86	.85	.84	.83	.82	.81
3.0	.78	.77	.76	.76	.75	.74	.73	.72	.71

TABLE 1—*Cont.*

Distance in Centimeters	Active Length in Centimeters								
	2.1	2.2	2.3	2.4	2.5	2.6	2.7	2.8	2.9
3.2	.70	.69	.69	.68	.67	.66	.66	.65	.64
3.4	.63	.62	.62	.61	.60	.60	.59	.58	.58
3.6	.56	.56	.56	.55	.54	.54	.53	.53	.52
3.8	.50	.50	.50	.49	.49	.49	.48	.48	.47
4.0	.45	.45	.45	.44	.44	.44	.43	.43	.43
4.5	.38	.38	.38	.37	.37	.37	.37	.37	.36
5.0	.31	.31	.30	.30	.30	.30	.299	.297	.295
5.5	.260	.259	.258	.257	.256	.255	.243	.253	.252
6.0	.216	.215	.215	.215	.215	.214	.213	.212	.211
6.5	.182	.182	.182	.182	.182	.182	.182	.182	.181
7.0	.162	.161	.161	.161	.161	.160	.160	.160	.160
7.5	.140	.140	.140	.140	.140	.140	.139	.139	.138
8.0	.125	.125	.125	.125	.125	.124	.124	.124	.123
	3.0	3.2	3.4	3.6	3.8	4.0	4.2	4.4	4.6
.3	12.0	11.2	10.5	9.8	9.3	8.8	8.4	8.0	7.6
.4	9.0	8.4	8.0	7.5	7.1	6.7	6.4	6.0	5.7
.5	7.1	6.7	6.4	6.0	5.7	5.4	5.1	4.9	4.6
.6	5.8	5.5	5.3	5.0	4.7	4.5	4.3	4.1	3.9
.7	4.9	4.7	4.4	4.2	4.0	3.8	3.6	3.5	3.3
.8	4.3	4.0	3.8	3.6	3.5	3.3	3.2	3.0	2.89
.9	3.7	3.5	3.3	3.2	3.0	2.85	2.75	2.63	2.54
1.0	3.3	3.1	2.95	2.80	2.68	2.56	2.45	2.35	2.26
1.2	2.60	2.50	2.37	2.26	2.16	2.08	2.00	1.92	1.85
1.4	2.12	2.03	1.95	1.86	1.80	1.73	1.66	1.60	1.55
1.6	1.78	1.70	1.62	1.57	1.51	1.45	1.40	1.35	1.31
1.8	1.53	1.46	1.40	1.35	1.30	1.25	1.22	1.17	1.14
2.0	1.34	1.29	1.24	1.19	1.15	1.10	1.07	1.04	1.00
2.2	1.15	1.12	1.08	1.04	1.00	.97	.94	.91	.88
2.4	1.00	.98	.95	.92	.89	.86	.84	.82	.79
2.6	.89	.86	.84	.82	.80	.77	.75	.73	.71
2.8	.80	.78	.76	.74	.72	.70	.68	.66	.64
3.0	.70	.68	.67	.66	.64	.62	.60	.59	.57
3.2	.64	.62	.60	.59	.57	.56	.55	.53	.52
3.4	.57	.56	.54	.53	.52	.51	.50	.49	.47
3.6	.52	.51	.50	.48	.47	.46	.45	.44	.43
3.8	.47	.46	.45	.44	.43	.42	.42	.41	.40
4.0	.42	.42	.41	.40	.40	.39	.38	.38	.37
4.5	.36	.36	.35	.35	.34	.34	.33	.32	.32
5.0	.295	.293	.290	.287	.284	.280	.276	.273	.269
5.5	.251	.250	.247	.244	.241	.238	.235	.232	.229
6.0	.210	.209	.207	.205	.203	.201	.200	.197	.195

TABLE I—*Cont.*

Distance in Centimeters	Active Length in Centimeters								
	3.0	3.2	3.4	3.6	3.8	4.0	4.2	4.4	4.6
6.5	.180	.180	.180	.179	.177	.176	.175	.173	.171
7.0	.160	.158	.157	.157	.155	.155	.153	.152	.151
7.5	.138	.138	.137	.136	.135	.134	.133	.132	.131
8.0	.123	.122	.121	.120	.120	.119	.118	.117	.116
	4.8	5.0	5.2	5.4	5.6	5.8	6.0	6.2	6.4
.3	7.2	6.9	6.6	6.3	6.0	5.7	5.4	5.2	5.0
.4	5.5	5.2	5.0	4.8	4.6	4.4	4.2	4.0	3.9
.5	4.4	4.3	4.1	3.9	3.8	3.6	3.5	3.4	3.3
.6	3.7	3.6	3.5	3.3	3.2	3.1	2.95	2.85	2.75
.7	3.2	3.1	2.95	2.85	2.75	2.65	2.56	2.47	2.40
.8	2.78	2.66	2.57	2.48	2.40	2.31	2.24	2.16	2.10
.9	2.45	2.35	2.27	2.19	2.11	2.05	1.98	1.92	1.87
1.0	2.18	2.10	2.03	1.96	1.90	1.85	1.79	1.73	1.67
1.2	1.78	1.71	1.65	1.60	1.55	1.50	1.45	1.41	1.37
1.4	1.49	1.44	1.39	1.34	1.30	1.26	1.23	1.19	1.16
1.6	1.26	1.22	1.18	1.15	1.12	1.09	1.06	1.03	.99
1.8	1.10	1.06	1.03	1.00	.93	.95	.92	.90	.87
2.0	.97	.94	.91	.88	.86	.84	.82	.80	.77
2.2	.86	.83	.81	.79	.77	.75	.73	.71	.69
2.4	.77	.75	.73	.71	.70	.68	.66	.64	.63
2.6	.69	.67	.66	.64	.62	.61	.59	.58	.56
2.8	.63	.61	.60	.58	.57	.55	.54	.53	.52
3.0	.56	.55	.53	.52	.51	.50	.49	.48	.47
3.2	.51	.50	.49	.48	.47	.46	.45	.44	.43
3.4	.46	.46	.45	.44	.43	.42	.41	.40	.40
3.6	.43	.42	.41	.40	.39	.39	.38	.37	.36
3.8	.39	.38	.38	.36	.35	.36	.35	.34	.34
4.0	.36	.36	.35	.34	.34	.33	.33	.32	.32
4.5	.31	.30	.30	.295	.290	.285	.280	.275	.270
5.0	.265	.261	.257	.253	.249	.245	.241	.237	.233
5.5	.226	.223	.220	.217	.214	.211	.208	.205	.202
6.0	.193	.190	.188	.186	.184	.182	.180	.177	.175
6.5	.170	.168	.166	.164	.162	.160	.158	.157	.155
7.0	.150	.148	.147	.146	.144	.143	.141	.140	.138
7.5	.130	.129	.128	.126	.125	.124	.123	.122	.120
8.0	.115	.115	.114	.113	.112	.111	.110	.109	.108
	6.6	6.8	7.0	7.2	7.4	7.6	7.8	8.0	8.5
.3	4.8	4.6	4.4	4.2	4.0	3.9	3.8	3.7	3.3
.4	3.8	3.6	3.5	3.4	3.2	3.1	3.0	2.95	2.75
.5	3.2	3.1	2.95	2.85	2.76	2.68	2.60	2.53	2.37

TABLE I—*Cont.*

Distance in Centimeters	Active Length in Centimeters								
	6.6	6.8	7.0	7.2	7.4	7.6	7.8	8.0	8.5
.6	2.66	2.58	2.50	2.42	2.36	2.28	2.21	2.15	2.02
.7	2.32	2.25	2.20	2.13	2.06	2.01	1.95	1.90	1.80
.8	2.02	1.97	1.91	1.85	1.81	1.76	1.72	1.67	1.58
.9	1.81	1.76	1.70	1.66	1.62	1.58	1.55	1.50	1.42
1.0	1.63	1.58	1.53	1.49	1.45	1.42	1.38	1.35	1.28
1.2	1.32	1.30	1.27	1.23	1.20	1.17	1.14	1.11	1.05
1.4	1.08	1.09	1.07	1.04	1.02	.99	.96	.94	.90
1.6	.97	.94	.92	.90	.88	.86	.84	.82	.77
1.8	.85	.83	.81	.79	.77	.75	.74	.72	.68
2.0	.75	.74	.72	.70	.69	.67	.66	.64	.61
2.2	.68	.66	.64	.63	.62	.60	.59	.57	.55
2.4	.61	.60	.59	.57	.56	.55	.54	.52	.50
2.6	.55	.54	.53	.52	.50	.49	.48	.47	.45
2.8	.50	.49	.48	.47	.46	.45	.44	.43	.41
3.0	.46	.45	.44	.43	.42	.41	.40	.40	.38
3.2	.42	.41	.40	.40	.39	.38	.37	.37	.35
3.4	.39	.38	.37	.37	.36	.35	.35	.34	.33
3.6	.36	.35	.35	.34	.33	.33	.32	.32	.31
3.8	.33	.33	.32	.32	.31	.30	.30	.295	.285
4.0	.31	.30	.30	.295	.291	.287	.283	.279	.267
4.5	.265	.260	.255	.251	.247	.243	.239	.235	.225
5.0	.229	.225	.222	.218	.215	.211	.208	.204	.196
5.5	.199	.196	.193	.190	.187	.185	.183	.181	.173
6.0	.173	.170	.168	.166	.164	.162	.160	.157	.152
6.5	.153	.151	.149	.147	.145	.144	.142	.140	.135
7.0	.136	.135	.133	.131	.130	.128	.127	.125	.121
7.5	.119	.118	.116	.115	.114	.113	.112	.110	.108
8.0	.107	.106	.105	.104	.103	.102	.101	.100	.097

Distance in Centimeters	9.0	9.5	10.0
.3	3.00	2.79	2.53
.4	2.55	2.40	2.23
.5	2.20	2.19	1.95
.6	1.88	1.78	1.67
.7	1.68	1.60	1.50
.8	1.49	1.40	1.33
.9	1.34	1.27	1.20
1.0	1.20	1.15	1.08
1.2	1.00	.94	.90
1.4	.84	.80	.76
1.6	.74	.70	.66
1.8	.64	.62	.59
2.0	.58	.55	.53

TABLE I—*Cont.*

Distance in Centimeters	Active Length in Centimeters		
	9.0	9.5	10.0
2.2	.52	.50	.47
2.4	.48	.46	.43
2.6	.43	.41	.39
2.8	.39	.38	.36
3.0	.36	.35	.33
3.2	.34	.32	.31
3.4	.31	.30	.290
3.6	.293	.284	.272
3.8	.274	.265	.255
4.0	.258	.250	.241
4.5	.215	.207	.199
5.0	.188	.181	.174
5.5	.167	.160	.154
6.0	.146	.140	.135
6.5	.130	.126	.122
7.0	.117	.114	.110
7.5	.105	.102	.098
8.0	.094	.091	.088

From the nomogram,

$$F_1 = 9.4;\ F_2 = 3.6$$
$$F_1 - F_2 = 9.4 - 3.6 = 5.8$$

Dose per milligram-hour from AB at

$$Q = \frac{5.8}{4 \times 2} = \frac{5.8}{8} = 0.73\ r_\gamma.$$

Example 1. For points on an end axis, there is only one "segment," i.e., the linear source itself.

$$F_1 = 8.9$$

Dose per milligram-hour at $N = \frac{8.9}{4 \times 2}$

$= 1.11\ r_\gamma$.

Example 2. For points on a central axis, the two segments are equal to each other and have the same F value.

$$F = 6.5$$
$$2F = 13.0$$

Dose per milligram-hour at $M = \frac{13.0}{4 \times 2}$

$= 1.63\ r_\gamma$.

For equivalent filtrations other than 0.5 mm. Pt, correction factors must be applied. These factors are given in Table II as percentages of the values calculated for 0.5 mm. Pt. The correction factor depends to some extent on the ratio of the distance to the active length. Two correction factors are therefore given for each filtration in order to cover the full range with an error less than 4 per cent.

When the linear source is inserted into

TABLE II

FILTRATION CORRECTION FACTORS

1 mm. Pt	1.5 mm. Pt	2.0 mm. Pt	2.5 mm. Pt
Ratio $\geq \frac{1}{4}$ 90%	Ratio $\geq \frac{1}{2}$ 80%	Ratio $\geq \frac{1}{2}$ 70%	Ratio $\geq \frac{1}{2}$ 64%
Ratio $< \frac{1}{4}$ 85%	Ratio $< \frac{1}{2}$ 75%	Ratio $< \frac{1}{2}$ 65%	Ratio $< \frac{1}{2}$ 57%

"Ratio" refers to the ratio of the perpendicular distance between the point and the source to the active length of the source.

a small volume of tissue, an additional correction must be made for tissue absorption and scattering. An approximate correction can be made by subtracting from the air dose 3 per cent for each centimeter of the distance.

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Twenty-eighth Annual Meeting: 1944, to be announced.

EDITORIAL

AN EVALUATION OF THE ROENTGEN METHODS USED IN MASS CHEST SURVEYS

IT IS generally recognized that the incidence of pulmonary tuberculosis might be effectively reduced if those individuals with active disease were isolated at the earliest possible moment. Since early isolation presupposes early detection and since the roentgen examination constitutes the most reliable method for the early diagnosis of pulmonary tuberculosis, it is evident that considerable progress in the control of this disease might be achieved if the population at large were examined roentgenographically at regular intervals. Until the early 1930's such a program was impractical due to the excessive cost of the roentgen methods then available. In recent years, however, this situation has been spectacularly changed by the development of the photofluorographic process in which a fluorescent image of the chest is photographed on films of reduced size (usually 4×5 inch, or 35 mm. film). Such films, when made in large numbers, are considerably less expensive to produce, process and handle than standard 14×17 inch roentgenograms. Recently, the roll paper process in which 14×17 inch roentgenograms are made on sensitized paper has been perfected and may also prove a useful method for the examination of large population groups.

The photofluorographic process has been used extensively in this country in the examination of inductees entering the armed services and of many thousands of civilians engaged in industry, and there is every reason to expect that the process will receive progressively wider application as time goes on. Before the outbreak of the current war, photofluorography was an established procedure in many foreign countries, particularly in Brazil and Scandinavia.

The diagnostic quality of photofluorographic and paper film is somewhat inferior to that of 14×17 inch roentgenograms and, as a result, many radiologists have become concerned lest the diagnostic error of these newer methods be high and thereby largely counteract their other advantages. In an effort to determine the relative merits of the various roentgen methods which are available for the examination of the chest (14×17 inch roentgenographic film, 4×5 inch photofluorographic film, 35 mm. photofluorographic film, 14×17 inch sensitized paper, and roentgenoscopy), clinical studies in which two or more of the methods were compared have been made by several investigators. The conclusions reached vary widely and seem to depend on the experience or inexperience, as the case may be, of the interpreter. Furthermore, many of these studies have not been carefully controlled from the viewpoint of selection of samples and statistical analysis and accordingly the overall picture tends to be confused.

A direct quantitative evaluation of the diagnostic quality of the various roentgen procedures may be made by a comprehensive investigation of the physical characteristics of the several methods. From such a study it is possible to list in unequivocal terms the relative detail with which roentgen images may be resolved by each method and to determine the smallest lesion which a given method is capable of recording. During the past two years such a study has been under way at the University of Chicago. An attempt has been made to correlate the various physical factors which

influence roentgenographic and roentgenoscopic quality so that the part played by each in the total effect may be correctly evaluated. Physiological considerations of viewing have also been included. Although this material will be presented in detail in a series of articles which will appear in forthcoming issues of this JOURNAL, it has been thought desirable to summarize here some of the preliminary findings of the study so far as they relate to the various roentgen methods used in the examination of the chest.

The resolving power of 14×17 inch roentgenograms is superior to that of all other roentgenographic materials. In order of merit below these films may be listed, 4×5 inch photofluorograms, when made with a stationary-focused grid; 14×17 inch sensitized paper; 35 mm. photofluorograms when made with a stationary-focused grid, and 4×5 inch photofluorograms, made without a grid; 35 mm. photofluorograms, made without a grid; and, finally, roentgenoscopy.

The low resolution provided by the roentgenoscopic method may be attributed to the low visual acuity of the eye when operating at the low intensity levels present under roentgenoscopic conditions. A grid improves the diagnostic quality of photofluorograms by reducing the amount of scattered radiation reaching the fluorescent screen, and thereby increasing the contrast of the roentgen images. Due to the high inherent contrast of 14×17 inch roentgenographic film, and the relatively short useful density range of 14×17 inch paper film, a grid is not useful when these materials are employed.

It must be realized that the above list represents conditions as of 1944, and undoubtedly it will require considerable revision from time to time in the years to come. Indeed, there is reason to expect that, before long, 4×5 inch photofluorograms and eventually 35 mm. photofluorograms will be equivalent to present-day 14×17 inch roentgenograms.

Although the foregoing discussion indicates the relative merits of the various roentgen procedures used in the examination of the chest, it does not provide any information as to the diagnostic error which may be expected from a given method in the detection of pulmonary tuberculosis. Physical studies reveal, however, that the smallest infiltrative lesion which may be detected by a competent observer using roentgenoscopy is between 0.5 and 1.0 cm. in diameter; smaller lesions, of course, can be detected with the other methods, with 14×17 inch roentgenograms being able to reproduce the smallest process. Since most significant lesions exceed 0.5 to 1.0 cm. in diameter, it is evident that all of the roentgen methods which are available for the examination of the chest should be capable of detecting the majority of tuberculous processes. One word of caution must be injected at this point, however. Since the resolution or detail with which a lesion may be seen varies widely in the different methods, one must not use the same diagnostic standards when interpreting the films produced by one method that are used when interpreting the films produced by another. For example, a particular tuberculous lesion will present considerably less detail in a 35 mm. film than that seen in a 14×17 inch roentgenogram. Indeed, if the lesion is small, its true significance may be easily overlooked if the roentgenologist unconsciously insists on the fulfillment of the criteria employed in the diagnosis of the larger film. A spectacular instance of this occurred recently when an acquaintance of the writer was asked to test his skill in interpreting 35 mm. photofluorograms. Fourteen by seventeen inch and 35 mm. films of several hundred individuals, of whom approximately 20 per cent were known to have minimal pulmonary tuberculosis, were submitted to this well known radiologist who until this time had read relatively few small films. The results of the test were at first highly disconcerting because a considerable number of cases called "positive" from the 14×17 inch films were called "negative" from the 35 mm. films. For-

tunately, however, the radiologist did not stop here but wisely reviewed the two series of films, comparing each 35 mm. photofluorogram with each 14×17 inch roentgenogram. In this way, he was able to establish for himself the diagnostic criteria which should be used in the interpretation of small films. After this educational program, another comparative series of 35 mm. photofluorograms and 14×17 inch roentgenograms was submitted to him for interpretation. The diagnostic error in reading the small films this time was of a much lower order.

The foregoing is a forceful demonstration that an ability to correctly diagnose the films produced by one roentgenographic method does not presuppose an equal ability to interpret the films produced by another. First, it is necessary for one to establish for himself the diagnostic criteria which are more or less peculiar to the roentgenograms made by a particular method. Such education may be accomplished in at least two ways. In one perspective may be gained by the examination of many thousands of films observed over a considerable period of time. This is the method which almost every roentgenologist employs during his training period. The trained roentgenologist, however, may follow a less tedious procedure. He may establish the diagnostic criteria which he is seeking by comparing the appearance of a representative number of tuberculous lesions produced on films with which he is familiar (for example, 14×17 inch roentgenograms) with those produced on films with which he is unfamiliar. Such a method was used by the roentgenologist referred to above.

In summarizing, it is evident that all of the newer roentgenological methods which are in use today can be considered acceptable from a diagnostic standpoint for use in mass roentgen surveys for tuberculosis control. It is extremely important, however, that the interpreter of the films clearly understands the diagnostic criteria of the roentgenographic process which is employed. Failure to do this will immediately introduce a large number of diagnostic errors.

R. H. M.

SOCIETY PROCEEDINGS, CORRESPONDENCE AND NEWS ITEMS

Items for this section solicited promptly after the events to which they refer.

MEETINGS OF ROENTGEN SOCIETIES*

UNITED STATES OF AMERICA

AMERICAN ROENTGEN RAY SOCIETY
Secretary, Dr. H. Dabney Kerr, University Hospital, Iowa City, Iowa. Annual meeting: Joint Meeting of American Roentgen Ray Society and Radiological Society of North America, Palmer House, Chicago, Ill., Sept. 24–29, 1944.

AMERICAN COLLEGE OF RADIOLOGY
Secretary, Mac F. Cahal, 540 N. Michigan Ave., Chicago, Ill. Annual Meeting: Stevens Hotel, 10:00 A.M., June 14, 1944.

SECTION ON RADIOLOGY, AMERICAN MEDICAL ASSOCIATION
Secretary, Dr. J. T. Murphy, 421 Michigan St., Toledo, Ohio. Annual meeting: Chicago, Ill., June 12–16, 1944.

ARKANSAS RADIOLOGICAL SOCIETY
Secretary, Dr. J. S. Wilson, Mack Wilson Hospital, Monticello, Ark. Meets every three months and also at time and place of State Medical Association.

RADIOLOGICAL SOCIETY OF NORTH AMERICA
Secretary, Dr. D. S. Childs, 607 Medical Arts Bldg., Syracuse, N. Y. Annual meeting: Joint Meeting of American Roentgen Ray Society and Radiological Society of North America, Palmer House, Chicago, Ill., Sept. 24–29, 1944.

RADIOLOGICAL SECTION, BALTIMORE MEDICAL SOCIETY
Secretary, Dr. Walter L. Kilby, Baltimore. Meets third Tuesday each month, September to May.

SECTION ON RADIOLOGY, CALIFORNIA MEDICAL ASSOCIATION
Secretary, Dr. Earl R. Miller, University of California Hospital, San Francisco, Calif.

RADIOLOGICAL SECTION, CONNECTICUT MEDICAL SOCIETY
Secretary, Dr. Max Climan, 242 Trumbull St., Hartford, Conn. Meets bi-monthly on second Thursday, at place selected by Secretary. Annual meeting in May.

SECTION ON RADIOLOGY, ILLINOIS STATE MEDICAL SOCIETY
Secretary, Dr. H. W. Ackemann, 321 W. State St., Rockford, Ill.

RADIOLOGICAL SECTION, LOS ANGELES COUNTY MEDICAL ASSOCIATION
Secretary, Dr. Roy W. Johnson, 1407 S. Hope St., Los Angeles, Calif. Meets on second Wednesday of each month at the County Society Building.

RADIOLOGICAL SECTION, SOUTHERN MEDICAL ASSOCIATION
Secretary, Dr. Roy G. Giles, Temple, Texas.

BROOKLYN ROENTGEN RAY SOCIETY
Secretary, Dr. Leo Harrington, 880 Ocean Ave., Brooklyn, N.Y. Meets monthly on fourth Tuesday, October to April.

BUFFALO RADIOLOGICAL SOCIETY
Secretary, Dr. Joseph S. Gian-Francheschi, 610 Niagara St., Buffalo, N. Y. Meets second Monday of each month except during summer months.

CHICAGO ROENTGEN SOCIETY
Secretary, Dr. F. H. Squire, 1754 W. Congress St., Chicago 12, Ill. Meets second Thursday of each month October to April inclusive at the Palmer House.

CINCINNATI RADIOLOGICAL SOCIETY
Secretary, Dr. Samuel Brown, 707 Race St., Cincinnati, Ohio. Meets third Tuesday of each month, October to May, inclusive.

CLEVELAND RADIOLOGICAL SOCIETY
Secretary, Dr. D. D. Brannan, 11311 Shaker Blvd., Cleveland 4, Ohio. Meets at 6:30 P.M. at Allerton Hotel on fourth Monday each month, October to April, inclusive.

DALLAS-FORT WORTH ROENTGEN STUDY CLUB
Secretary, Dr. X. R. Hyde, Medical Arts Bldg., Fort Worth, Texas. Meetings held in Dallas on odd months and in Fort Worth on even months, on third Monday, at 7:30 P.M.

DENVER RADIOLOGICAL CLUB
Secretary, Dr. Edward J. Meister, 366 Metropolitan Bldg., Denver, Colo. Meets third Friday of each month at Denver Athletic Club.

DETROIT ROENTGEN RAY AND RADIUM SOCIETY
Secretary, Dr. E. R. Witwer, Harper Hospital. Meets monthly on first Thursday from October to May, at Wayne County Medical Society Building.

FLORIDA RADIOLOGICAL SOCIETY
Acting Secretary, Dr. Walter A. Weed, 204 Exchange Bldg., Orlando, Fla. Meetings in May and November.

GEORGIA RADIOLOGICAL SOCIETY
Secretary, Dr. James J. Clark, 478 Peachtree St., Atlanta, Ga. Meets in November and at annual meeting of Medical Association of Georgia in the spring.

RADIOLOGICAL SOCIETY OF KANSAS CITY
Secretary, Dr. Arthur B. Smith, 800 Argyle Bldg., Kansas City, Mo. Meets third Thursday of each month at a place designated by the president.

ILLINOIS RADIOLOGICAL SOCIETY
Secretary, Dr. Wm. DeHollander, St. John's Hospital, Springfield, Ill. Meets three times a year.

INDIANA ROENTGEN SOCIETY
Secretary, Dr. H. C. Ochsner, Methodist Hospital, Indianapolis. Meeting held the second Sunday in May annually.

KENTUCKY RADIOLOGICAL SOCIETY
Secretary, Dr. W. C. Martin, 321 W. Broadway, Louisville. Meets annually in Louisville on first Saturday in Apr.

LONG ISLAND RADIOLOGICAL SOCIETY
Secretary, Dr. Marcus Wiener, 1430-48th St., Brooklyn, N. Y. Meets Kings County Med Soc. Bldg. monthly on fourth Thursday, October to May, 8:30 P.M.

LOUISIANA RADIOLOGICAL SOCIETY
Secretary, Dr. J. R. Anderson, 1130 Louisiana Ave., Shreveport. Meets annually during Louisiana State Medical Society Meeting.

MICHIGAN ASSOCIATION OF ROENTGENOLOGISTS
Secretary, Dr. E. M. Shebesta, 1429 David Whitney Bldg., Detroit. Three meetings a year, Fall, Winter, Spring.

MILWAUKEE ROENTGEN RAY SOCIETY
Secretary, Dr. C. A. H. Fortier, 231 W. Wisconsin Ave., Milwaukee, Wis. Meets monthly on second Monday at University Club.

MINNESOTA RADIOLOGICAL SOCIETY
Secretary, Dr. Annette T. Stenstrom, 1218 Medical Arts Bldg., Minneapolis, Minn. One meeting a year at time of Minnesota State Medical Association.

NEBRASKA RADIOLOGICAL SOCIETY
Secretary, Dr. D. A. Dowell, Medical Arts Bldg., Omaha, Nebr. Meets third Wednesday of each month, at 6 P.M. at either Omaha or Lincoln.

NEW ENGLAND ROENTGEN RAY SOCIETY
Secretary, Dr. Hugh F. Hare, Lahey Clinic, Boston, Mass. Meets monthly on third Friday, Boston Medical Library.

RADIOLOGICAL SOCIETY OF NEW JERSEY
Secretary, Dr. H. R. Brindle, 501 Grand Ave., Asbury Pk. Meets annually at time and place of State Medical Society. Mid-year meetings at place chosen by president.

NEW YORK ROENTGEN SOCIETY
Secretary, Dr. Ramsay Spillman, 115 East 61st St., New York City. Meets monthly on third Monday, New York Academy of Medicine, at 8:00 P.M.

NORTH CAROLINA ROENTGEN RAY SOCIETY
Secretary, Dr. Major Fleming, Rocky Mount, N. C. Annual meeting at time and place of State Medical Society. Mid-year scientific meeting at place designated.

*Secretaries of Societies not here listed are requested to send the necessary information to the Editor.

NORTH DAKOTA RADIOLOGICAL SOCIETY
Secretary, Dr. L. A. Nash, St. John's Hospital, Fargo. Meetings held by announcement.

CENTRAL NEW YORK ROENTGEN RAY SOCIETY
Secretary, Dr. C. F. Potter, 820 S. Crouse Ave., Syracuse. Three meetings a year. January, May, November.

OHIO RADIOLOGICAL SOCIETY
Secretary, Dr. J. E. McCarthy, 707 Race St., Cincinnati. Meets at time and place of annual meeting of Ohio State Medical Association.

PACIFIC ROENTGEN SOCIETY
Secretary, Dr. L. H. Garland, 450 Sutter St., San Francisco, Calif. Meets annually, during meeting of California Medical Association.

PENNSYLVANIA RADIOLOGICAL SOCIETY
Secretary, Dr. L. E. Wurster, 416 Pine St., Williamsport. Annual meeting at Penn Alto Hotel, Altoona, Pa., May 13–14, 1944.

PHILADELPHIA ROENTGEN RAY SOCIETY
Secretary, Dr. R. P. Barden, University Hospital. Meetings first Thursday of each month from October to May inclusive at 8:15 P.M., in Thompson Hall, College of Physicians, 19 S. 22d St.

PITTSBURGH ROENTGEN SOCIETY
Secretary, Dr. R. G. Alley, 4800 Friendship Ave. Meets second Wednesday each month, 4:30 P.M., October to June, Pittsburgh Academy of Medicine.

ROCHESTER ROENTGEN RAY SOCIETY, ROCHESTER, N. Y.
Secretary, Dr. Sidney Larson, Strong Memorial Hospital. Meets monthly on third Monday from October to May, inclusive, 8 P.M. at Strong Memorial Hospital.

ROCKY MOUNTAIN RADIOLOGICAL SOCIETY
Secretary, Dr. A. M. Popma, 220 N. First St., Boise, Idaho.

ST. LOUIS SOCIETY OF RADIOLOGISTS
Secretary, Dr. E. W. Spinzig, 2646 Potomac, St. Louis, Mo. Meets fourth Wednesday of each month, except June, July, August, and September, at a place designated by the president.

SAN DIEGO ROENTGEN SOCIETY
Secretary, Dr. Henry L. Jaffe, Naval Hospital, Balboa Park, San Diego, Calif. Meets monthly on first Wednesday at dinner.

SAN FRANCISCO RADIOLOGICAL SOCIETY
Secretary, Dr. Martha Mottram, 450 Sutter St., San Francisco. Meets monthly on third Thursday at 7:45 P.M., first six months of year at Toland Hall, University of California Hospital, second six months at Lane Hall, Stanford University Hospital.

SHREVEPORT RADIOLOGICAL CLUB
Secretary, Dr. R. W. Cooper, Charity Hospital, Shreveport, La. Meets monthly on third Wednesday, at 7:30 P.M., September to May inclusive.

SOUTH CAROLINA X-RAY SOCIETY
Secretary, Dr. T. A. Pitts, Baptist Hospital, Columbia, S. C. Meets in Charleston on first Thursday in November, also at the time and place of South Carolina State Medical Association.

TENNESSEE RADIOLOGICAL SOCIETY
Secretary, Dr. J. M. Frere, 707 Walnut St., Chattanooga, Tenn. Meets annually at the time and place of the Tennessee State Medical Association.

TEXAS RADIOLOGICAL SOCIETY
Secretary, Dr. Herman Klapproth, Sherman, Texas.

UNIVERSITY OF MICHIGAN DEPARTMENT OF ROENTGENOLOGY STAFF MEETING
Meets each Monday evening from September to June, at 7 P.M. at University Hospital.

UNIVERSITY OF WISCONSIN RADIOLOGICAL CONFERENCE
Secretary, Dr. E. A. Pohle, 1300 University Ave., Madison, Wis. Meets every Thursday from 4:00–5:00 P.M., Room 301, Service Memorial Institute.

VIRGINIA RADIOLOGICAL SOCIETY
Secretary, Dr. E. L. Flanagan, 116 E. Franklin St., Richmond, Va. Meets annually in October.

WASHINGTON STATE RADIOLOGICAL SOCIETY
Secretary, Dr. Thomas Carlile, 1115 Terry St., Seattle. Meets fourth Monday each month, October through May, College Club, Seattle.

X-RAY STUDY CLUB OF SAN FRANCISCO
Secretary, Dr. J. M. Robinson, University of California Hospital. Meets monthly in evening on third Thursday.

CUBA

SOCIEDAD DE RADIOLOGÍA Y FISIOTERAPIA DE CUBA
President, Dr. J. Manuel Viamonte, Hospital Mercedes, Habana, Cuba. Meets monthly in Habana.

BRITISH EMPIRE

BRITISH INSTITUTE OF RADIOLOGY INCORPORATED WITH THE RÖNTGEN SOCIETY
Medical Members' meeting held monthly on third Friday at 2:30 P.M. and Ordinary Meeting at same time on following Saturday, October to May, 32 Welbeck St., London, W.1.

SECTION OF RADIOLOGY OF THE ROYAL SOCIETY OF MEDICINE (CONFINED TO MEDICAL MEMBERS)
Meets on the third Friday of each month at 4:15 P.M. at the Royal Society of Medicine 1, Wimpole St., London, W. 1.

FACULTY OF RADIOLOGISTS
Secretary, Dr. M. H. Jupe, 32 Welbeck St., London, W. 1 England.

SECTION OF RADIOLOGY AND MEDICAL ELECTRICITY, AUSTRALASIAN MEDICAL CONGRESS
Secretary, Dr. H. M. Cutler, 139 Macquarie St., Sydney, New South Wales.

RADIOLOGICAL SECTION OF THE VICTORIAN BRANCH OF THE BRITISH MEDICAL ASSOCIATION
Secretary, Dr. Keith Hallam, St. George's Hospital, K.E.W., Melbourne, E. 4, Victoria, Australia. Meets monthly from March to November inclusive.

CANADIAN ASSOCIATION OF RADIOLOGISTS
Secretary, Dr. A. D. Irvine, 540 Tegler Bldg., Edmonton, Alberta.

SECTION OF RADIOLOGY, CANADIAN MEDICAL ASSOCIATION
Secretary, Dr. C. M. Jones, Inglis St., Ext., Halifax, N. S.

RADIOLOGICAL SECTION, NEW ZEALAND BRITISH MEDICAL ASSOCIATION
Secretary, Dr. Colin Anderson, Invercargill, New Zealand. Meets annually.

SOUTH AMERICA

SOCIEDAD ARGENTINA DE RADIOLOGIA
Secretary, Dr. Guido Gotta, Buenos Aires, Argentina. Meetings are held monthly.

CONTINENTAL EUROPE

SOCIEDAD ESPANOLA DE RADIOLOGIA Y ELECTROLOGIA
Secretary, Dr. J. Martin-Crespo, Fuencarral, 7, Madrid, Spain. Meets monthly in Madrid.

SOCIÉTÉ SUISSE DE RADIOLOGIE (SCHWEIZERISCHE RÖNTGEN-GESELLSCHAFT)
Secretary for French language, Dr. A. Grosjean La Chaux de Fonds.
Secretary for German language, Dr. Scheurer, Metzgasse Biel. Meets annually in different cities.

SOCIETATEA ROMANA DE RADIOLOGIE SI ELECTROLOGIE
Secretary, Dr. Oscar Meller, Str. Banul Maracine, 30, S. I., Bucaresti, Roumania. Meets second Monday in every month with the exception of July and August.

ALL-RUSSIAN ROENTGEN RAY ASSOCIATION, LENINGRAD: USSR in the State Institute of Roentgenology and Radiology, 6 Roentgen St.
Secretaries, Drs. S. A. Reinberg and S. G. Simonson. Meets annually.

LENINGRAD ROENTGEN RAY SOCIETY
Secretaries, Drs. S. G. Simonson and G. A. Gasterin. Meets monthly, first Monday at 8 o'clock, State Institute of Roentgenology and Radiology, Leningrad.

MOSCOW ROENTGEN RAY SOCIETY
Secretaries, Drs. L. L. Holst, A. W. Ssamygin and S. T. Konobejevsky. Meets monthly, first Monday, 8 P.M.

SCANDINAVIAN ROENTGEN SOCIETIES
The Scandinavian roentgen societies have formed a joint association called the Northern Association for Medical Radiology, meeting every second year in the different countries belonging to the Association.

JOINT MEETING OF THE AMERICAN ROENTGEN RAY SOCIETY AND THE RADIOLOGICAL SOCIETY OF NORTH AMERICA

Attention is again called to the Joint Meeting of the American Roentgen Ray Society and the Radiological Society of North America which is to be held at the Palmer House, Chicago, Illinois, September 24 to 29, 1944. Plans are progressing satisfactorily and a very successful meeting is foreseen. Committees have been appointed which are looking after all of the details in connection with the meeting.

The Coordinating Committee consists of Dr. L. C. Kinney, 1831 Fourth Avenue, San Diego 1, California, Dr. E. R. Witwer, Harper Hospital, Detroit 1, Michigan, and Dr. E. L. Jenkinson, St. Luke's Hospital, Chicago, Illinois. Titles and abstracts of papers should be sent to Dr. Kinney or Dr. Witwer before July 1, 1944.

Dr. Lawrence Reynolds, 110 Professional Building, Detroit 1, Michigan, is Chairman of the Committee on Commercial Exhibits. Those who have not already taken space in the exhibit and are interested in doing so should communicate with Dr. Reynolds at an early date.

The Committee on Scientific Exhibits of which Dr. Clarence E. Hufford, 421 Michigan Street, Toledo, Ohio, is Chairman anticipates assembling an instructive and interesting exhibit.

Dr. Warren W. Furey, 6844 Oglesby Avenue, Chicago, Illinois, is in charge of the Refresher Courses which will begin on Sunday, September 24. These are always an important part of a meeting and it is certain that this year they will prove of more than ordinary interest and educational value.

The complete program of the meeting will be published in a future issue of the JOURNAL. In the meantime it is hoped that radiologists will make their plans to attend and that those who wish to take an active part in the meeting will communicate as soon as possible with those in charge of the arrangements.

RADIOLOGICAL SOCIETY OF NEW JERSEY

At the annual meeting of the Radiological Society of New Jersey held in Atlantic City on April 26, 1944, the following officers were elected for the ensuing year: *President*, Dr. J. H. Wyatt; *Vice President*, Dr. H. J. Perlberg; *Secretary*, Dr. H. R. Brindle; *Treasurer*, Dr. W. H. Seward; *Counsellor*, Dr. W. O. Wuester.

AMERICAN BOARD OF RADIOLOGY

The American Board of Radiology will conduct examinations on September 22, 23, and 24, 1944, at the Palmer House, Chicago, Illinois. Those wishing to be examined at that time must have their application on file by August 1, 1944.

B. R. KIRKLIN
Secretary

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ABSTRACTS OF ROENTGEN AND RADIUM LITERATURE

ROENTGEN DIAGNOSIS

GENITOURINARY SYSTEM

SWEETSER, T. H. Retroperitoneal tumors influencing the kidneys and ureters. *J. Urol.*, May, 1942, *47*, 619–631.

The advent of intravenous and retrograde urography, exposing films in anteroposterior and lateral projections, has helped in the diagnosis of retroperitoneal tumors, but the diagnosis may still be difficult. The authors present 8 case reports to illustrate some of the difficulties.

The first case was a sixteen year old boy with an abdominal mass and hematuria. A retrograde pyelogram showed upward displacement of the right kidney and mesial displacement of the right ureter; operation revealed a retroperitoneal neuro-epithelioma. The second case was that of a twenty-four year old woman with an obvious perinephric abscess. The sinus tract did not heal following drainage and a retrograde pyelogram showed the presence of the abscess and a hydronephrosis. Compression of the ureter was present, as well as forward and lateral displacement. This led to the impression of a retroperitoneal mass as the etiological factor in the whole picture. Operation revealed an undifferentiated, very malignant tumor compressing the ureter.

The third case was a boy of fifteen who complained of recurrent abdominal pain, and swelling. Roentgenographic studies showed a retroperitoneal tumor, containing calcium, which was displacing the kidney downward and the diaphragm upward. The stomach was displaced forward and to the right. A high diastolic blood pressure and periodic headaches suggested an adrenal medullary tumor. Operation disclosed an adrenal paraganglioma. The roentgen findings might have been simulated by a cyst of the tail of the pancreas.

The fourth case was that of a sixty-six year old man who complained of lumbar pain, frequency and noctusia. An abdominal mass could be felt. The right ureter was displaced mesially. Operation revealed an adenocarcinoma of the colon.

Case 5 was a twenty year old woman who complained of right flank pain. An intravenous urogram showed outward and downward displacement of the right kidney. The left ureter showed slight lateral displacement. The spleen was enlarged and several discrete cervical nodes were palpable. The diagnosis was Hodgkin's disease and the abdominal mass was due to enlarged abdominal lymph nodes. A simple renal cyst was present which added to the confusing picture and raised the question of a primary renal tumor with metastasis to the lymph nodes.

The next 2 cases showed displacement of the kidneys and ureters from enlarged lymph nodes due to metastases from primary tumors in the testicle and ureter respectively. The eighth case had a mass overlying the promontory of the sacrum which proved to be a hydronephrotic ectopic kidney.

In conclusion, roentgenography has proved to be the greatest single aid in diagnosing these confusing cases.—*R. M. Harvey.*

PRINCE, C. L. Malignant tumors of the spermatic cord; a brief review with presentation of a case of angio-endothelioma. *J. Urol.*, June, 1942, *47*, 793–799.

This case is the first angio-endothelioma of the spermatic cord and the seventy-fifth malignant tumor of the cord to be reported. The patient was a white male, aged sixty-three, who had had a progressive swelling of the scrotum for fourteen months. Growth had been rapid for three months. Physical examination revealed a mass arising from the upper pole of the testis which did not transmit light. A roentgenogram showed no calcification.

At operation the contents of the left scrotum were removed en masse and a tumor of the spermatic cord was found which histologically proved to be an angio-endothelioma. Postoperative roentgen therapy was given. Twenty-seven months later the patient returned with apparent orbital metastases which responded readily to roentgen therapy.

Seventy per cent of tumors of the spermatic cord are benign, according to Thompson; 71 of the reported 74 malignant tumors were sarcoma and only 3 carcinoma. The classification of tumors of the spermatic cord as proposed by

Hinman and Gilison is given by the author. They divide the tumors both benign and malignant into three groups: epithelial, mesoblastic and heterologous. The dermoids are included in the benign heterologous group and the teratomas in the malignant heterologous group. The angio-endotheliomata, such as reported in this article by Prince, are supposed to arise in cells lining vascular and lymphatic channels. This should serve to put these tumors in the sarcomatous group because of mesoblastic origin.

The differential diagnosis of spermatic cord tumors must include hernia, hydrocele, hematocele, spermatocele and testicular tumors.

The treatment of a malignant cord tumor is thorough removal of the tumor, testis, epididymis and as much of the cord as possible followed by postoperative deep roentgen therapy. —*R. M. Harvey.*

JEWETT, HUGH J., SLOAN, LOUISE L., and STRONG, GEORGE H. Does vitamin A deficiency exist in clinical urolithiasis? *J. Am. M. Ass.*, Feb. 20, 1943, *121*, 566–569.

This investigation consisted in a clinical study of 20 patients and a microscopic examination of 78 autopsy cases with urinary calculi. In the clinical group the authors attempted to select reliable visual tests for vitamin A deficiency and have supplemented these subjective tests with the determinations of the level of vitamin A in the blood. In obtaining data, 20 patients with urolithiasis in the Brady Urological Institute and 40 normal subjects comprising a control group were given a series of tests. Determinations of the concentrations of vitamin A in the blood plasma were made in the 20 cases of urolithiasis and in 33 normal controls.

In summary, the writers say that in none of these 98 cases was there any evidence of vitamin A deficiency. Although a vitamin A free diet produces widespread epithelial changes which may lead to the formation of urinary calculi in experimental animals, there is no positive proof that a "subclinical" vitamin A deficiency is an etiologic factor in urolithiasis in man.—*S. G. Henderson.*

THOMPSON, G. J., and GREENE, L. F. Ureterocele; a clinical study and a report of 37 cases. *J. Urol.*, June, 1942, *47*, 800–809.

Excretory urography rarely demonstrates a ureterocele although it will reveal the ureterectasis and pyelectasis which may accompany it. Most authors believe ureterocele to be congenital in origin. Gottlieb believes that there are two causative factors—an abnormally narrow ureteral orifice and a weakness of Waldeyer's sheath.

Other congential deformities are frequently associated with ureterocele which supports the congenital theory of their etiology. Some cases are undoubtedly of congenital origin, and some of acquired origin.

Two-thirds of the authors' cases occurred between the ages of thirty and fifty. The symptoms are inconstant, with pain the most frequent. Other symptoms are hematuria, frequency, dysuria, etc. The cystoscopic picture may be confusing in the presence of associated inflammation and edema.

In excretory urograms a ureterocele appears as a spherical or sausage-shaped filling defect in the bladder near the mouth of the ureter. Occasionally a surrounding halo is present. Stones may be present in the ureterocele. The authors, in a series of 21 cases studied by excretory urography, were able to make a diagnosis of ureterocele in only 4 cases on the urogram alone. The most common roentgen finding was an irregular dilatation of the lower third of the ureter which tapered to a point at the ureteral orifice. Although they did not find the excretory urogram of special value in making a specific diagnosis of ureterocele, the authors were able to demonstrate other congenital anomalies of the urinary tract, to estimate kidney function, to note secondary obstructive nropathies and to show stones within the ureteroceles.

Treatment is not necessary in the absence of symptoms. Dilatation of the ureteral orifice may be of temporary benefit. The most satisfactory form of treatment is ureteroneatotomy. The results of treatment are entirely satisfactory.—*R. M. Harvey.*

KRETSCHMER, HERMAN L. Stone in the ureter; clinical data based on 500 cases. *Surg., Gynec. & Obst.*, June, 1942, *74*, 1065–1077.

This paper is based on a study of 500 unselected patients with stone in the ureter, covering a period from 1917 to 1941.

Sex. Of the 500 patients studied, 400 were males and 100 females.

Age. Stone in the ureter is rare at the extremes of life. Stone occurred with the greatest frequency in the third, fourth and fifth decades.

Location. The stone was located on the right side in 229 patients, on the left side in 259 and on both sides in 12.

The Stone. (1) Number of stones: In the largest number of instances the symptoms are due to the presence of a single stone, 89.4 per cent of the present series. Multiple stones may vary greatly in number from 2 to 100 or more. (2) Size: Fortunately, most stones in the ureter are small. (3) Chemical composition of stones: 102 stones were analyzed; in only 9 cases was only one element present. Calcium phosphate was the predominant element in 45 stones and calcium oxalate in 35.

Symptoms and Signs.

1. Pain was the first symptom in 88 per cent of the cases. It occurred at some time during the illness in 93.6 per cent. The pain usually radiates downward along the course of the ureter to the internal or external genitalia or upward into the renal area. The pain is usually very severe.

2. Hematuria may be the first and only symptom of which the patient complains. Hematuria was present in 34.2 per cent at some time during the illness and in 4.6 per cent as the first symptom noted.

3. Nausea and vomiting occurred in 46.2 per cent. Previous passage of stones was elicited in the history of 14.2 per cent of these patients.

4. Other symptoms. Backache was present in 37.2 per cent. Frequency of urination was noted in 42 per cent and burning in 30.4 per cent. Thirty-six per cent stated that pus had previously been found in the urine. Chills and fever were present in 17.6 per cent.

The Examination.

1. Urinalysis. The findings were positive in 83.43 per cent and negative in 15.56 per cent. The results of the urinary examinations were as follows: albumin in 24.4 per cent; sugar in 0.8 per cent; red blood cells in 43 per cent; white blood cells in 69.2 per cent; casts in 7.6 per cent. The importance of repeated examinations cannot be overemphasized.

2. Culture studies of the urine. Cultures were made in 385 patients of which 34.54 per cent were positive and 65.43 per cent were sterile.

3. Blood pressure studies. These were normal except in patients in the older age group.

4. Roentgen-ray observations. This begins with a plain roentgenogram. Positive reports were obtained in 95.79 per cent and negative reports in 4.21 per cent. The presence of a shadow along the course of the ureter warrants further investigation to determine whether one is dealing with a stone or with an extra-ureteral shadow. This can be done by the use of a shadowgraph catheter with a double exposure film and shift in the tube. In the case of very stout patients it is advantageous to use two films.

5. Intravenous urography. In most cases it reveals dilatation of the ureter and of the kidney pelvis. Stone in the ureter is probably the most frequent cause of hydronephrosis. In some instances the urogram fails to visualize the outline of the pelvis and ureter while an increased density of the kidney outline is shown. This is probably due to a temporary inhibition of function.

6. Retrograde movement of stone. Because of this possibility a roentgenogram should be made on each patient just before operation. Retrograde movement of the stone occurred in 10 patients in the present series.

Treatment. (1) One hundred and twenty-eight patients passed their stones without treatment; 196, with manipulation; and 114 were operated on. (2) Dangers of too frequent manipulation are stressed by the author. (3) A policy of "watchful waiting" in the average case is still desirable.—*Mary Frances Vastine.*

HIEMSTRA, W., and CREEVY, C. D. Tumors of the urinary bladder. *Radiology*, August, 1942, *39*, 175–183.

This article is a report of 174 cases of tumor of the bladder treated at the University Hospitals, University of Minnesota Medical School from January 1, 1930 to December 31, 1939. They included 28 benign papillomas and 146 carcinomas, 57 of which were papillary (39 per cent) and 89 infiltrating (61 per cent). Most of the carcinomas were too advanced to permit of radical operation. Tables and graphs are given showing the details of the treatment and results. Cases without metastases showed a five year survival rate of 15.7 per cent and those with metastases only 5.3 per cent. Among 70 cases of the infiltrating type the five year survival rate was only 4.2 per cent.

In the carcinoma cases the average duration of symptoms before reporting for treatment was 2.3 years. This should be materially reduced by educating the public in regard to the early symptoms of carcinoma of the bladder and by bringing about better cooperation between the public and the physicians. Hematuria is the commonest first symptom and the public does not realize its seriousness. They should be

taught to report for examination on the appearance of the slightest degree of hematuria. Increased frequency and dysuria were the next most frequent symptoms, being present in about one-third of the cases. Nocturia was present in about 15 per cent. Other symptoms occasionally seen were incontinence, a sense of incomplete emptying of the bladder, pain in the bladder region and renal colic.

External irradiation has not proved as effective as was hoped. In some cases the results may be improved by the careful use of radon and radium therapy. Contact and possibly supervoltage therapy may prove valuable.—*Audrey G. Morgan.*

NERVOUS SYSTEM

THOMPSON, RALPH M. Neuroblastoma (sympathoblastoma or neurocytoma) of the suprarenal medulla. *Ann. Int. Med.*, June, 1942, *16*, 1206–1220.

There are three types of tumor derived from the sympathetic nervous system of the suprarenal medulla: neuroblastoma, ganglioneuroma and paraganglioma (chromaffin cell tumors). The author considers only the first type which occurs almost entirely in infants and young children and occasionally in young adults.

He describes 3 cases in patients seventeen months, five years and eighteen years of age. Photomicrographs of the findings are given. The younger the patient, the less differentiated the cells in these tumors and the greater the malignancy. The patients generally grow progressively worse and die. In these cases the periods of hospitalization were 40, 191 and 105 days respectively.

Pepper has claimed that these tumors metastasize only to the liver and Hutchinson that they metastasize to the bones, chiefly those of the skull. The clinical symptoms are the same in the two groups. Frew claims that tumors of the left suprarenal metastasize to bones through the blood and those of the right suprarenal to the liver through the lymphatics.

The diagnosis depends largely on the metastatic lesions and the presence of an abdominal tumor.

Contrary to generally accepted opinion, these tumors are radiosensitive and life may be prolonged by irradiation. In the second case of this series a total dose of 4,500 r was given and the abdominal tumor shrank from the size of a child's head to a nodule 3×1.5×1.5 cm. in size found on autopsy. There is no doubt that life was prolonged by the irradiation but autopsy showed marked aplasia or destruction of bone marrow.

One case has been reported in which a patient was alive fifteen years after operation but in general surgery is not successful in these cases.—*Audrey G. Morgan.*

HAUSER, EMIL D. W. Neurofibroma (neurinoma) of the foot. *J. Am. M. Ass.*, April 10, 1943, *121*, 1217–1219.

Hauser reports the case of an eleven year old girl who had pain in the ball of the right foot for six months. Roentgenograms did not aid in diagnosis. At operation a neurinoma (neurofibroma) which was found attached to the plantar fascia was removed. Six months later the pain recurred. At the second operation a large sausage-shaped neurinoma (neurofibroma) was found beneath the plantar fascia and removed. The tumor was easily dissected from the tendon sheaths in the area and was traced up until it ended in the deep plantar nerve.

Ewing states that this type of tumor affects mainly the larger nerve trunks and occurs in nearly all parts of the body, forming encapsulated, slowly growing, firm or soft masses attached to the nerve trunk. They have a definite tendency to recur locally from remaining tumor tissue if not completely removed and may become malignant and locally invasive following recurrence.—*S. G. Henderson.*

SKELETAL SYSTEM

KLEINBERG, SAMUEL, and BUCHMAN, M. S. Spondylolisthesis; report of 3 cases in adults with forward displacement of the vertebra below the level of the laminar defect. *J. Bone & Joint Surg.*, Oct., 1942, *24*, 899–906.

The authors state that it has been the consensus that the essential factor or *sine qua non* in spondylolisthesis is a bilateral defect separating the posterior arch from the body of the vertebra. Moreover, according to all records, the slipping always occurred in the vertebra with the laminar defect. They report 3 cases which they consider unusual for the following reasons:

1. The vertebra which became displaced was not the one with the laminar defect but the one below it.

2. In 1 of the cases, the laminar defect was apparently unilateral, the subluxation occurring with the apophyseal joint of the uninjured side as the center of rotation.

3. In each instance, there was a trauma adequate to produce a fracture of the laminae; the symptoms appeared after the injury, and hence it seems possible that the slipping resulted from a laminar fracture—that is, that the spondylolisthesis was of traumatic origin.

4. Although the patients were adults, the slipping in each instance was only mild.

5. In 2 of the patients, the laminar defects appeared identical in location and extent, and there was a similar displacement and rotation of the posterior arches.

The authors remark that it remains for future experience to determine whether these findings necessarily indicate that the laminar defect is congenital, and militate against the suggestion that the spondylolisthesis is of traumatic origin. —*R. S. Bromer.*

LYON, ERNST. Intercalary bones of the intervertebral disc. *J. Bone & Joint Surg.*, Oct., 1942, *24*, 805–811.

Lyon defines the intercalary bone of the intervertebral disc as an osseous formation completely surrounded by the tissue of the intervertebral disc; that is, it is interposed between the two vertebral bodies. Intercalary bones are irregularly shaped, isolated osseous formations and are usually found in the anterior peripheral portion of the disc. They occur in older individuals who suffer from spondylosis and are found mainly in the discs of the lumbar region, although they are occasionally encountered in the thoracic and cervical portions of the spinal column.

On roentgenographic examination, the posteroanterior view reveals irregularly outlined, small or larger shadows in the intervertebral space, in the neighborhood of the vertebral corner. They are usually situated between two opposite spondylotic osteophytes. The lateral view shows them as homogeneous, triangular shadows, the size of a rice kernel or even larger, situated in the anterior portion between the upper and lower edges of two adjacent bodies. They sometimes touch the edge of the vertebral body and may approach two opposite spondylotic osteophytes. An open space usually appears between these shadows and the vertebral surface.

Considering the high incidence of spondylosis, these bones provide a rare accidental finding of only secondary importance. Symptoms, if any are present, are not characteristic of the condition. There are no particular symptoms definitely connected with the presence of the intercalary formations. They do not occur as the sequel to an accident and they represent the ultimate stage of a curative process in a lesion of the outer portion of the disc and of the nucleus pulposus, which has been present for a considerable time.—*R. S. Bromer.*

BAKER, LENOX D. Rhizomelic spondylosis; orthopaedic and roentgen therapy. *J. Bone & Joint Surg.*, Oct., 1942, *24*, 827–830.

Baker in this report describes the apparatus and routine used in 22 cases of rhizomelic spondylosis treated by roentgen therapy combined with well established orthopedic measures. The therapy was directed at alleviating pain, relaxing muscle spasm, and correcting deformities, with the hope of arresting the disease. Baker employed the principles outlined by Swaim which consisted of gradual hyperextension of the spine, relaxation of muscles, corrective exercises to straighten the spine and increase chest expansion, and immobilization of the spine to hold the correction. Swaim believed that the trauma of use plus continued muscle spasm served to keep the disease active, and to keep the involved tissues in an inflamed condition during convalescence. He used plaster of paris on leather body jackets to obtain complete immobilization which he did not believe was accomplished by the popular type of spinal braces.

Baker found that in a majority of patients severe pain was usually present during the early period of treatment. The addition of roentgen therapy gave an early alleviation of pain with relief of muscle spasm and it allowed rapid correction of deformities and more vigorous attempts at restoration of muscle balance. The roentgen therapy, if not contraindicated, was given over either the entire spine or the involved area, depending on the clinical findings. Two hundred kilovolts were used in treating areas of about 5 by 15 cm; 150 r was given over one or more areas daily for three to five treatments; if necessary, the dosage was repeated after three to six weeks.

The average length of hospitalization with the combined therapy was ten days. The cor-

rection of deformities, thus far, has been satisfactory. In the cases treated, follow-up roentgenograms showed no increased calcification in the spinal ligaments and the patients as a whole maintained the increase which had been obtained in the range of motion. The most satisfactory results were obtained in the early cases but surprisingly good improvement was noted in some of the patients in whom marked deformity and advanced calcification were present.—*R. S. Bromer.*

HART, VERNON L. Primary genetic dysplasia of the hip with and without classical dislocation. *J. Bone & Joint Surg.*, 1942, *24*, 753–771.

Classical congenital dislocation of the hip is secondary to a primary genetic dysplasia of the hip, or flat acetabulum, and develops during intra-uterine life, or in the course of the first or second year of postnatal life. Dislocation is a consequence of the primary dysplasia or "flat socket" it is a secondary and incidental phenomenon. It has been difficult to explain genetically congenital hip dislocation. An individual does not inherit the condition but does inherit a primary defect, or hip dysplasia, which produces anatomical and physiological alterations of the joint which is called the "flat socket." To make a true genetic investigation of hip dislocation, roentgenographic consanguinity studies are essential according to Hart's opinion. In his investigation he found it cannot be sex linked because it is observed to pass from father to son. There is no "dislocation" gene but there is a "hip dysplasia" gene. The gene involved is not a recessive gene but is a dominant one.

Hart believes that dysplasia of the hip without dislocation is a distinct clinical and roentgenographic entity, and should be included in textbooks in the chapter on classical dislocation, because the two entities have the same genetic etiology. Dysplasia of the hip without dislocation occurs more frequently and may be more disabling than classical dislocation. Acclivity of the roof of the acetabulum is the primary anatomical feature of a dysplastic hip joint. Hip dysplasia refers not only to the acetabulum, but also to all joint-forming parts of the hip joint. Primary hip dysplasia can lead much more easily to complete dislocation in the female than in the male. The condition with dislocation on one side is frequently associated with dysplasia without dislocation on the opposite side. It may remain asymptomatic for many years; in some individuals it may never be shown clinically. The extreme disability observed in some individuals is caused by traumatic arthritis of the hip joint resulting from mechanical factors of instability and incongruity.

Hart emphasizes proper roentgenographic technique to prevent distorted views and erroneous measurements of the hip joint. The patient must be relaxed and placed flat on the back. The lower extremities should be in contact, the hips extended, and the patellae facing directly forward. The roentgen tube must be centered in the midline of the body and directly over the superior border of the symphysis pubis. Roentgenographic diagnosis of hip dysplasia without dislocation is very difficult during the first months of life, and the condition is rarely recognized except where the routine examinations of the newborn are made. Most often it is dislocation of the opposite hip which leads to its discovery.

The following hip joint lesions may at times require careful study for differentiation from primary hip dysplasia: coxa plana or Legg-Perthes' disease, epiphyseal separation, tuberculosis, infectious arthritis, hypertrophic arthritis and aseptic necrosis. To these should also be added: late septic hip joint, neoplasm, neurotrophic lesions, old fracture-dislocations, and endocrine dysfunction.

The article is well illustrated with roentgenograms and the treatment is described in detail. The patient may be relieved of pain and disability by conservative treatment and surgical measures.—*R. S. Bromer.*

COMPERE, EDWARD L., and WALLACE, GEORGE. Etiology of aseptic necrosis of the head of the femur after transcervical fracture. *J. Bone & Joint Surg.*, Oct., 1942, *24*, 831–841.

The incidence of non-union resulting from treatment of intracapsular fractures of the hip by methods of internal fixation has been found to be very high. The primary etiological factor in aseptic necrosis is disruption of the blood supply to the head of the femur. The development of techniques for obtaining satisfactory lateral roentgenograms of the hip has undoubtedly resulted in more accurate reduction of transcervical fractures. The authors planned an experiment to try to determine whether or not immediate reduction and complete immobilization by internal fixation of the fragments would

influence favorably the incidence of aseptic necrosis of the head of the femur.

In the experiments carried out, necrosis with collapse of the weight-bearing portion of the cortex of the head occurred in 2 of 6 cases following accurate reduction of the fracture and pinning of the fragments, although the fracture united. In these 2 cases the head of the femur may have been viable when union occurred but, with partially depleted blood supply, the minimal traumata of continued use and weight bearing may have resulted in the necrosis and disintegration.

Compere and Wallace conclude that necrosis of the head of the femur occurs less often if the fracture of the transcervical neck is immediately and accurately reduced and adequately immobilized. The prognosis for survival of the head of the femur is definitely less satisfactory if the reduction of the fracture is not anatomically correct or if pinning of the fragments is not adequate for complete immobilization

They found that death of the head of the femur when the fracture was neither reduced nor immobilized occurred in 9 of the 12 hips studied. It is possible, they believe, that weight-bearing activity and friction between the fragments may have ruptured the blood vessels that were left intact after fracture in a portion of the posterior capsule. However, this theory does not explain the difference in incidence of necrosis in cases in which the head was pinned without accurate reduction and in those in which the head was pinned in an anatomically correct position.

The union of a fracture of the neck of the femur, regardless of the method of reduction or of immobilization, does not constitute of itself an end-result. The condition of each head of the femur should be studied at intervals for at least five years after union and beginning weight bearing by those surgeons who are qualified by training and experience to recognize roentgenographic changes and to interpret them in terms of pathological processes such as aseptic necrosis.—*R. S. Bromer.*

HEFKE, HANS W., and TURNER, VERNON C. The obturator sign as the earliest roentgenographic sign in the diagnosis of septic arthritis and tuberculosis of the hip. *J. Bone & Joint Surg.*, Oct., 1942, *24*, 857–869.

In the anteroposterior roentgenographic examination of the pelvis and hip, a soft tissue shadow may be seen just medial to the acetabulum on the inner aspect of the pelvis. The border of this shadow ordinarily starts from a point lateral to and below the lower pole of the sacroiliac joint, follows the line of the pelvic inlet downward over the acetabulum, and disappears behind the upper ramus of the pubis. This shadow in normal children measures from 2 to 8 mm. at its widest point. Usually the shadow on one side corresponds in width to the shadow on the other. There may be, however, a minimal difference in width of this shadow in normal individuals. It has been noted that certain changes occur in the contour of this shadow in cases of septic arthritis and in tuberculosis of the hip. The authors' purpose in their study reported in this paper was to determine the anatomical basis of this shadow and its importance in the early roentgenographic diagnosis of pathological conditions about the hip.

Hefke and Turner believe that the obturator internus muscle has been definitely proved to be the substratum of the shadow. The fibers of the obturator internus were laid bare in a postmortem specimen, and a wire was placed along its medial border. This corresponded exactly to the position of the border of the shadow as seen in all the roentgenograms. In regard to the pathological changes underlying the variation of the shadow in septic arthritis, the tendon of the obturator internus passes immediately adjacent to the capsule of the hip, and extension of the inflammatory process may well lead to a roentgenographically visible swelling of the obturator muscle.

In a small number of cases it was noted that the obturator shadow was obscured. After making the study the conclusion was reached that an obscured obturator shadow is a positive finding indicative of pathological changes in the hip joint. They also believe the widening, the changes in contour, and the obscurity of the border, may be compared with the well known changes of the psoas shadow seen in roentgenographic examination of a psoas abscess, perinephritic abscess, or other inflammatory process overlying, or in contact with, the psoas muscle. They advise the use of the usual technique for routine roentgenograms of the pelvis. No special advantage was shown in the use of soft tissue technique. Care must be taken that the pelvis is level or one obturator may be thrown into higher relief than the other one. They found that in some cases it was advantageous to obtain an anteroposterior view of the pelvis with the hips in marked abduction, flexion to 90

degrees, and marked external rotation (so-called "frog position," frequently used in obtaining lateral views of the hip joint) which seemed to bring the obturator shadow into greater relief.

They conclude that in certain types of pathological conditions of the hip joint the obturator shadow becomes obscured, or it becomes widened with a more curved border. The greatest value of the sign is in early roentgenographic diagnosis of septic hip disease. It is of some importance in early diagnosis of tuberculosis of the hip. It was not present in fractures about the hip, osteochondritis, slipping of the capital epiphysis, rheumatic fever with clinical involvement of the hip, osteomyelitis of the pelvis not involving or neighboring the hip joint and soft tissue infection about the thigh with marked inguinal lymphadenopathy.—*R. S. Bromer.*

WEAVER, JAMES B. Calcification and ossification of the menisci. *J. Bone & Joint Surg.*, Oct. 1942 *24*, 873–882.

Weaver reports 2 cases of calcification of the menisci of the knee joint. In addition to his 2 cases, he found reports of 76 others in the literature. Primary or non-traumatic and secondary or traumatic calcifications are two separate and distinct entities that have little in common except the presence of calcium. The primary type occurs in older individuals, the secondary in younger. There was a preponderance of males in the series of cases in both types. Both menisci of both knees were affected in cases of the primary type, but only one meniscus in each knee was affected in cases of the secondary type.

In the primary cases, the calcium is laid down in parallel layers starting at the periphery of the cartilage and involving the entire length. The meniscus is smooth. The secondary cases show a localized zone of calcification, usually in one end or the other, and of the central type. This calcium piles up, causing a roughening and enlargement of the part of the cartilage affected.

In the roentgenogram, the primary type shows a waferlike aspect of the cartilage in the anteroposterior view and wedge-shaped shadows at each end of the meniscus in the lateral view. This suggests a diffuse calcification of all four menisci, although these areas may vary in size and density according to the age of the lesion. Calcification may be revealed in individual menisci at various times in the same patient. The roentgenographic appearance is characteristic and diagnostic. The secondary type shows a localized piling up of calcium, rough in outline, which might be confused with a joint mouse or ossification in the cartilage. The picture is not diagnostic.

In many instances primary calcification produces no symptoms. In some cases, symptoms such as pain, swelling, limitation of motion and occasionally hydrops, are severe enough to necessitate removal of the cartilage. Pain, swelling and limitation of motion are the principal symptoms of the secondary type. Hypertrophic arthritis, both in the affected joints and in other joints, was almost a constant finding in the primary cases.

Weaver states that in the primary type, the treatment is symptomatic. Excision of cartilage is infrequently necessary. Spontaneous cures were numerous in the reports in the literature. Extirpation is always necessary in the secondary type.

Ossification of the semilunar cartilage is a condition closely allied to calcification of the cartilage and may be a sequela to calcification. The symptoms, roentgenographic appearance, and treatment of secondary ossification are practically the same as in secondary calcification. Weaver found no evidence that primary calcification is a precursor of secondary ossification. He is of the opinion that many cases of secondary calcification do not progress to ossification. The cases of secondary ossification reported by Wollenberg, Eck, and Weaver are unquestionably true examples of heterotopic bone formation, and the other cases reported are probably in the same category.—*R. S. Bromer.*

FREUD, PAUL, and SLOBODY, LAWRENCE B. Symphalangism; a familial malformation. *Am. J. Dis. Child.*, April, 1943, *65*, 550–557.

Symphalangism results from failure of an interphalangeal joint to form. There may be complete or partial lack of development of the joint. When the interphalangeal joint is entirely absent, there is no dividing line between the two phalanges. They appear, rather, as a single long phalanx with a common bone marrow cavity. The trabecular structure of the bones is changed to conform to the new stresses of pull and pressure. When the interphalangeal joint is developed slightly, there are two distinct bones and two bone marrow cavities. The ligaments are shorter than normal and there is a solid fibrous connection between the two bones. Motion is impossible. When the development of the interphalangeal joint has progressed a little further

some bending and stretching become possible. In the family reported in this paper, the family tree covered four generations. Nine of 10 members of the family were known to have symphalangism. This illustrates the dominant character of the inherited malformation. The roentgenograms which are reproduced in the paper illustrate varying degrees of the condition and demonstrate the manner in which the various stages of articular differentiation occur during the life of the embryo. The earliest change is a constriction of the phalangeal beam. This progresses until only a central bony bridge remains. This bridging becomes less distinct and then invisible, and then a normal joint space is formed. The process may be stopped at any point before complete differentiation with resulting symphalangism.—*R. S. Bromer.*

Stone, Samuel. Osteomyelitis of the long bones in the newborn. *Am. J. Dis. Child.*, Oct., 1942, *64*, 680–688.

There are few reports of osteomyelitis of the long bones in the newborn. Osteomyelitis in the newborn child appears to be a benign disease; but when it is a manifestation of sepsis, the prognosis is more grave and depends on the severity of the septic process. Stone reports 4 cases in which the striking feature was the benign course of the disease. Characteristic were the quick healing of the osseous lesions, as observed in the roentgenograms, the rapidity of subperiosteal new bone formation, the short duration of discharge from sinuses and the quick healing of the operative wounds. In none of his patients did sequestration occur. None of his infants was acutely ill, and the admission to the hospital was usually sought because of localized swellings of the extremities or diminished motion of the arms and legs rather than because of severe systemic symptoms. The general condition of the infants remained good; they took their feedings well and gained weight. The operative procedures were limited to incision and drainage of palpable localized abscesses without involving the bone. Green and Shannon ascribed the benign course of osteomyelitis in infants to the peculiar anatomy of the bone at this age. According to them, the vascular spaces of the bone are larger and the bone is of spongy texture and less rigid than in older children. The thin cortical bone at the metaphysis allows free communication between the marrow and the subperiosteal spaces and thus provides a mechanism for decompression of pus from the metaphysis and the marrow to the subperiosteal spaces. The periosteum is more loosely attached in infants, and this further aids decompression. Rupture of the periosteum, with decompression of pus, also makes for absence of gross sequestration. The problem in treatment, as with osteomyelitis in older children, is to tide the patient over the period of bacteriemia. The treatment of the osseous lesion is of secondary importance.—*R. S. Bromer*

Barber, C. Glenn. Osteochondrosis deformans tibiae; nonrachitic bow leg in children. *Am. J. Dis. Child.*, Nov., 1942, *64*, 831–842.

Barber states that at the time of writing the most common cause of development of bow leg during childhood in his community is osteochondrosis of the medial tibial condyle. He believes that the reason the condition has not been more widely recognized is due to the fact that it has been described by a diversity of names in previous reports. Since Blount called attention to it as a cause of acute bending of the legs in children, he has encountered no less than a dozen cases in which the deformity might be rightfully attributed to osteochondrotic changes in the tibial epiphyses. Although the children had received adequate amounts of vitamin D and although other evidence of rickets was wanting, the parents had either been told or were of the opinion that the deformities of the legs were due to that disease.

The roentgenographic and pathologic changes are like those of coxa plana and those in the many bones similarly involved elsewhere but are quite different from those of rickets. When the tibial condyles are involved, two distinct types occur. These two types are dependent on the age at which the condition becomes manifest, and they have been designated as the infantile and adolescent types of the disease denominated by the less cumbersome though more grossly descriptive term "tibia vara."

In cases of infantile type a history of normal development, except for some overweight, for one or two years is usually obtained. The deformity occurs bilaterally and when mild may disappear on one or both sides spontaneously. When it is unilateral there is a limp in walking, and when it is bilateral there is a waddle. When the deformity occurs during infancy, a bulbous enlargement of the medial tibial condyle is palpable on physical examination and visible in the

roentgenogram. This beaklike projection at the metaphysis has been shown to contain islands of cartilage. Faulty growth of the upper tibial epiphyseal cartilage and delayed ossification of its medial half result in a wedge-shaped deformity of the epiphysis. The lower tibial epiphyseal cartilage occasionally shows similar though less pronounced changes; if this occurs, some lateral bowing just above the ankle may appear. Elsewhere the tibial shaft is perfectly straight.

The onset of the adolescent type is between the ages of six and twelve years in previously normal children. The deformity usually occurs on one side only. The adolescent type looks different in the roentgenogram. It is an arrest of growth rather than a dysplasia. The roentgenographic appearance of the infantile type gradually changes to that of the adolescent type, so that the two can be distinguished later only by the history. Recurvation at the knee and relative flatfoot are present, irrespective of the age.

The treatment of osteochondrosis deformans tibiae depends on the stage at which it is instituted. Measures to prevent further deformity or to correct existing deformity, even when extreme, have proved effective during the plastic stage of the condition. When deformity is disabling or unsightly after the osteochondrosis has become arrested, tibial osteotomy is advisable. However, if it is performed before arrest is assured, recurrence of the deformity is to be expected.—*R. S. Bromer.*

BRAKELEY, ELIZABETH. Late rickets. *Am. J. Dis. Child.*, Feb., 1943, *65*, 314–319.

A case of late rickets in a girl aged eleven is reported. Active rickets after two years of age is uncommon, and only a few cases have been reported of its occurrence in older children who have been extremely resistant to treatment and who required massive doses of vitamin D before cure was accomplished. In the case reported in this article, healing began after three months, following a daily dose of 40,000 U. S. P. units of vitamin D. The child continued to show extreme decalcification of bones, a high serum calcium level, a low level of serum phosphorus and a high serum phosphatase level for fifteen months after healing of the rickets. It was believed that she had a fairly high degree of hyperparathyroidism secondary to the rickets, although no parathyroid glands were found on operation. Gradual improvement followed irradiation of the neck in the region of the parathyroid by the same dosage twenty-three days later to the left side of the neck. It is impossible to prove, however, whether the gradual increase in the calcification of the shafts of the bones and the return to normal values of serum calcium, phosphorus and phosphatase were a result of the irradiation or would have occurred without further treatment.—*R. S. Bromer.*

BLOOD AND LYMPH SYSTEM

CHAMBERLAIN, FRANCIS L. Diseases of the lesser circulation. *Radiology*, August, 1942, *39*, 151–152.

Regulation of the lesser circulation differs from that of the greater circulation and these differences must be known in order to understand diseases of the lesser circulation. The most important abnormal condition in the lesser circulation is hypertension, which is most frequently caused by failure of the left ventricle. This causes engorgement from backing up of the blood in the pulmonary circuit. If the right ventricle fails at the same time this engorgement does not occur. Other less frequent causes of pulmonary hypertension or engorgement are mitral stenosis, and congenital, traumatic or syphilitic left-to-right heart shunts.

Pulmonary hypertension is usually associated with pulmonary engorgement because of the large diameter of the pulmonary capillaries. This explains the variability in the roentgenogram of the lung in the common types of heart disease. It also explains why enlargement of the heart must take place before pulmonary hypertension with its attendant dyspnea and other symptoms can occur. It explains the increased density and size of the hilar shadows, increased lung markings and the radiopaque appearance of the lung fields proper.

Diseases of the lesser circulation characterized by intrinsic or extrinsic obstruction of the pulmonary vessels (cor pulmonale) may be acute, subacute or chronic. The clinical signs may differ from those of pulmonary hypertension due to the relative absence of pulmonary engorgement. There may be no rales and the lung fields may be unusually radiolucent. But underlying lung disease may confuse the picture. Cyanosis is usually more marked and in the chronic types the fingers and toes may be-

come clubbed and polycythemia may develop.—*Audrey G. Morgan.*

STEINBERG, M. F., GRISHMAN, A., and SUSSMAN, M. L. The angiocardiographic demonstration of an arteriovenous fistula. *Surg., Gynec. & Obst.*, July, 1942, *75*, 93–96.

The roentgen demonstration of peripheral arteriovenous fistulas has been accomplished by the introduction of radiopaque substances through direct arterial punctures. A simpler method is available through the application of the angiocardiographic technique. This consists of the rapid introduction of diodrast 70 per cent through a special needle into an antecubital vein, making serial roentgen exposures which may be recorded either fluorographically or roentgenographically.

A case is described in which the clinical picture was unmistakably that of an arteriovenous fistula of the subclavian or axillary vessels. However, more accurate localization of its site was considered desirable before operative intervention because the surgical approach for a fistula between the subclavian vessels would be transthoracic while that for a fisutla between the axillary vessels would be entirely extrathoracic. Two injections of 30 cc. of diodrast 70 per cent were given; the first into the left antecubital vein, the second into the right antecubital vein. Roentgenograms were taken rapidly with the roentgen tube centered over the supraclavicular region, shoulder, and upper arm. Injection of the involved side definitely established the identity and size of the left axillary, subclavian and innominate veins and the axillary artery. It was evident that the opaque material must have reached the axillary vein directly from the axillary artery at a point near their origins, since both of these vessels were simultaneously visualized. It was likely that the actual fistula was present at a point proximal to the origin of the axillary vessels. The third portion of the subclavian artery was therefore designated as the probable site of the fistula.—*Mary Frances Vastine.*

WATSON, JAMES R., LICHTY, J. M., HILL, J. M., and MILLER, R. B. The use of venograms for the localization and study of arteriovenous fistula. *Surg., Gynec. & Obst.*, June, 1943, *76*, 659–664.

Venograms were performed on 3 patients with arteriovenous fistulas. These cases are reported in the article.

In traumatic arteriovenous fistula, the changes in the vein consist of hypertrophy and dilatation. The dilatation may be diffuse and extend for some distance proximal to the level of the fistula or it may be maximal locally at the site of the fistula. As long as the valves remain competent, no changes are apt to occur in the venous trunk distal to the fistula. Dilatation in the distal portion is uncommon for the arterial blood, taking the course of least resistance, passes through the fistula and into the proximal vein. The essentially normal status of the involved vein distal to the level of the fistula makes it readily adaptable for venography.

Indications for Venography.

1. Demonstration of fistulas at or proximal to the roots of the extremities where injection of the artery (arteriogram) above the level of the fistula would be difficult or even impossible because of its anatomical position.

2. Venography might be used in conjunction with arteriography when the presence of multiple fistulas is suspected.

3. In cases in which arteriography or venography could be used, venography has the advantage of permitting the use of media which are inert when injected into the venous circulation and which are readily excreted by the kidneys.

In their summary, the authors include the following statements:

1. Studies of the venograms in each instance showed the vein distal to the fistula to be normal.

2. The venograms demonstrated a marked difference in the collateral venous circulation between 1 case in which the vein had been ligated distal to the fistula at the time of injury for control of hemorrhage and 2 cases in which this had not been necessary.

3. Ligation of the vein distal to the fistula appears to have the same beneficial effect on the extremity that ligation proximal to the fistula has on the heart.—*Mary Frances Vastine.*

ROENTGEN AND RADIUM THERAPY

WEED, L. A., ECHTERNACHT, A. P., MEISTER, E. J., and ISENHOUR, ROGER. The effect of therapeutic doses of x-ray on infections and inflammations; experimental studies. *Surg., Gynec. & Obst.*, Aug., 1942, *75*, 157–160.

Part of the data for this paper was obtained at the State University of Iowa and the remainder at the Indiana University Medical Center. The summary and conclusions reached are:

(1) In guinea pigs roentgen irradiation had no beneficial effect in the treatment of gas gangrene due to *Clostridium welchii*.

(2) Roentgen irradiation was ineffective in reducing the number of viable vegetative forms of a two hour culture of *Clostridium welchii*.

(3) Roentgen irradiation was ineffective in retarding the rate of growth of *Clostridium welchii*.

(4) Roentgen irradiation did not prevent the development of hemolysin or dermonecrotic toxin of *Clostridium welchii*.

(5) Roentgen irradiation would not prevent the development of dermonecrosis even if used before or immediately after the injection of *Clostridium welchii* toxin.

(6) Roentgen irradiation would not inactivate either the hemolysin or dermonecrotic toxin when applied in vitro.

(7) Roentgen irradiation would not inactivate in vitro either the hemolysin or dermonecrotic toxin of *Staphylococcus aureus*.

(8) Roentgen irradiation would not inactivate in vitro the toxin of *Corynebacterium diphtheriae*.

In no experiment has any real effect been demonstrated with doses of roentgen rays even beyond that permitted for human therapy.—*Mary Frances Vastine.*

Medical Uses of Radium; summary of reports from experimental research centers for 1940. Edited by the Joint Radiology Committee of the Medical Research Council and the British Empire Cancer Campaign. *Brit. J. Radiol.*, Feb., 1942, *15*, 56–62.

This is a continuation of the series of articles on Medical Uses of Radium which has been issued by the Medical Research Council for many years. It describes the work of the Radium Beam Therapy Research Team in London, including a study of roentgen-ray and gamma-ray effects. The construction of the pressure-insulated two-million volt electrostatic generator is proceeding satisfactorily and further experiments have been made on the properties of moulded bakelite-graphite mixtures to which traces of heavy elements have been added. A mixture containing calcium fluoride has proved better than the ones previously used. The Royal Cancer Hospital in London reports work on the spatial distribution of radiation during radium treatment. The Mount Vernon Hospital of Northwood reports a method of measuring very small partial discharges of a condenser dosimeter. The Strangeways Research Laboratory in Cambridge reports generalizations on the biological actions of radiation, the inactivation of plant viruses by radiation, the production of chromosome abnormalities in irradiated plant material, the effect of gamma rays on normal embryonic cells *in vivo* and the quantitative biological analysis of human biopsies. The Department of Medicine of the University of Cambridge reports on the measurement of the effects of roentgen and gamma irradiation on normal and malignant cells by means of ultraviolet photomicrography. Dr. Gray and Dr. Read of the Mount Vernon Hospital, Northwood, report on the biological effects of ionizing radiations, the summation of neutron and gamma-ray effects, the neutron irradiation of mouse tumors *in vivo* and *in vitro* and the lethal effect of alpha radiation on bean roots. The Barnato Joel Laboratories of the Middlesex Hospital, London, report on the effects of irradiation on rabbits' ovaries using radon instead of radium.—*Audrey G. Morgan.*

Wilson, C. W. Method of "dose-finding" for combinations of rectangular radiation fields. *Brit. J. Radiol.*, May, 1942, *15*, 145–149.

The methods of dose-finding heretofore in use have applied only to circular fields. In this article a method of representing the three-dimensional distribution of radiation throughout rectangular radiation fields is described. For each roentgen-ray field the dose contours are determined by measurements in a pressdwood phantom in a number of planes containing the central ray, at various angles to one of the major axes of the field. The measurements are made with a Siemens dosage-rate meter. The apparatus is illustrated and dose contours in various planes are given.—*Audrey G. Morgan.*

Levitt, W. M. Discussion of the constitutional effects of radiation, with special reference to volume dose. *Brit. J. Radiol.*, April, 1942, *15*, 99–103.

This was a discussion by the Faculty of Radiologists with Dr. Levitt presiding as chairman. He discussed the term "volume dose" which means the relationship between the total amount of radiation energy absorbed and the constitutional effects produced. A "dose" of radiation does not mean a definite quantity. A dose may be given that has such serious constitutional effects that it more than nullifies the good local effect. Volume dose cannot always be calculated accurately but progress has been made in this respect. Even if the volume dose

is accurately calculated there are other circumstances to be taken into consideration, such as the effect on the blood-forming tissues and the decreased toleration to radiation in liver disease.

Dr. Levitt then introduced Prof. Maynecord who discussed the relationship between the roentgen and energy absorption per unit mass of air and tissues. He recommended the use of the megagram-roentgen as a unit, as the gram-roentgen is far too small. The megagram-roentgen is the energy absorption if a uniform dose of 1,000 roentgens were given throughout 1,000 grams of air or soft tissues. He also discussed the energy absorption in the beam for different techniques and the relationship of total energy absorption to protection and its dependence on wave length. The chemical effects of roentgen rays were discussed and a method of calculation of optimum penetration given.—*Audrey G. Morgan.*

SMITHERS, D. W., ELLIS, FRANK, and GRIMMETT, L. G. Discussion on the constitutional effects of radiation, with special reference to volume dose. *Brit. J. Radiol.*, May, 1942, *15*, 141–144.

Dr. Smithers in a meeting of the Faculty of Radiologists discussed the complexity of the task of measuring the total amount of radiation energy absorbed by the body. The clinical problem is much more complex than the physical one and even the physicists have had difficulty in measuring integral dosage. The integral dose, measured in megagram-roentgens, is a far more valuable index of the treatment given than the surface dose. He gives a table showing the integral doses and the tumor doses in two groups of cancers, one of the pharynx and larynx and one of the stomach and esophagus; the tumor doses were the same in the two groups but the integral doses were more than twice as large in one group as in the other.

He also discussed the use of vitamin C in association with irradiation and the apparent good results of such treatment, and the use of histaminase in the treatment of roentgen sickness.

Dr. Ellis said that the volume dose of total energy absorbed is given by the product of the area of the field, the surface dose, a graph reading and the ratio of the time to deliver the dose to the field concerned, divided by that to deliver the same dose to a field at the center of which the scattering is maximal. However, there are inaccuracies in this and all other methods and an accurate estimation of total energy absorbed is as yet impossible.

It has been found that the fatal volume dose per kilogram in a rat is about the same as that of a single dose of roentgen rays to the whole human body. This suggests that some toxic substance may be produced by irradiation that has the same quantitative action on man and animals.

Dr Grimmett described a method of measuring the total energy absorbed by the human body by the use of a life-size celluloid man in which the sum of the ionization currents in the air-gaps between the celluloid plates is measured. It offers a quick method of measuring energy absorption for gamma rays and high voltage roentgen rays.—*Audrey G. Morgan.*

APTHOMAS, INEZ. Contact x-ray treatment of cavernous angioma in children. *Brit. J. Radiol.*, Feb., 1942, *15*, 43–46.

Mention is made of an article in the AMERICAN JOURNAL OF ROENTGENOLOGY AND RADIUM THERAPY, November, 1939, which reviews cases of the above-named disease treated from 1933–1936. This article brings the record of the treated cases up to date and describes a new method of treatment used in the Holt Radium Institute, Manchester, England, since 1936. The new method is that of contact roentgen therapy, using a Chaoul tube at 60 kv. and 4 ma. The applicators range from 1.5 cm. in diameter at 15 cm. focal skin distance to 9 cm. in diameter at 10 cm. focal skin distance. The smaller ones are usually adequate.

This treatment is used only in cavernous angioma proper and solid hemangioma. The capillary type, or port-wine stain, is almost completely radioresistant. For the average lesion a dose of 400 r is sufficient, repeated at intervals of two months. Tables are given showing the results for the radium methods used before 1937 and the contact roentgen therapy cases treated since then. There were about 92 per cent cures for both methods, the other 8 per cent being improved. The contact therapy takes less time than the elastoplast method; no anesthetic is needed and it is never necessary to remove an unsightly gold seed. Radium implants are to be preferred in very bulky lesions. The number of treatments required varies with the dose and the size of the lesion.—*Audrey G. Morgan.*

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